Supplementary Figures

Supplementary Figure 1. INPP4B is frequently upregulated in colon cancers. Quantitation of INPP4B mRNA expression levels in laser capture micro-dissected CRC cells relative to paired adjacent noncancerous colon tissues using qPCR. The relative abundance of INPP4B mRNA in each noncancerous colon tissue was arbitrarily designated as 1; the expression level of INPP4B mRNA in the paired colon cancer was presented as the fold of increase.
Supplementary Figure 2. INPP4B promotes proliferation and survival of colon cancer cells.

(A) Whole cell lysates from Lim1215 and EB colon cancer cells stably transduced with the control shRNA (shControl) or two individual INPP4B shRNAs (shINPP4B1 and shINPP4B2) were subjected to Western blot analysis. Data are representative of three individual experiments.

(B) WiDr and HCT116 cells stably transduced with the control shRNA (shControl) or INPP4B shRNA (shINPP4B1) were treated with EGF (100nM) for 15 minutes. Whole cell lysates were subjected to Western blot analysis. Data are representative of three individual experiments.

(C) WiDr, HCT116, Lim1215 and EB cells were transduced with the control shRNA (shControl) or two individual INPP4B shRNAs (shINPP4B1 and shINPP4B2). Forty-eight hours later, cells were subjected to CellTiter-Glo assays. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(D) Lim1215 and EB cells were transduced with the control shRNA (shControl) or two individual INPP4B shRNAs (shINPP4B1 and shINPP4B2). Forty-eight hours later, cells were subjected to BrdU incorporation assays. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(E) Quantitation of colony formation as shown in Figure 2D. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(F) SW620, HT-29, and MDA-MB-231 cells transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) were subjected to BrdU incorporation assays. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(G) Quantitation of colony formation as shown in Figure 2H. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.
Supplementary Figure 3. Akt and SGK3 cooperatively regulate colon cancer cell proliferation downstream of INPP4B.

(A) Whole cell lysates from WiDr, HCT116, Lim1215, and EB cells stably transduced with the control shRNA (shControl) or two individual INPP4B shRNAs (shINPP4B1 and shINPP4B2) were subjected to Western blot analysis. The data shown are representative of three individual experiments.

(B) HCT116 cells stably transduced with shINPP4B1 were transduced with the vector alone, myr-SGK3 cDNA, myr-Akt cDNA, or myr-SGK3 cDNA plus myr-Akt cDNA. Forty-eight hours later, cells were subjected to BrdU incorporation assays. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(C) WiDr and HCT116 cells stably transduced with shControl or shINPP4B1 were transduced with the vector alone, myr-SGK3 cDNA, myr-Akt cDNA, or myr-SGK3 cDNA plus myr-Akt cDNA. Forty-eight hours later, whole cell lysates were subjected to Western blot analysis. The data shown are representative of three individual experiments.
Supplementary Figure 4. Activation of Akt and SGK3 is positively associated with the expression of INPP4B, whereas the expression of PTEN is negatively related to INPP4B expression in colon cancers in vivo.

(A) Representative microphotographs of IHC staining of INPP4B, pSer473-Akt, Akt, pThr320-SGK3, SGK3, and PTEN on FFPF colon cancer sections from the cohort of 124 colon cancers detailed in Supplementary Table 1. Scale bar, 100µm.

(B) Regression analysis of the relation between INPP4B and pSer473-Akt expression levels in the cohort of 124 colon cancers.

(C) Regression analysis of the relation between INPP4B and pThr320-SGK3 expression levels in the cohort of 124 colon cancers.

(D) Comparison of the levels of PTEN between colon cancers with high expression of INPP4B and those with low expression defined using the median of INPP4B levels as the cutoff in the cohort of 124 colon cancers (colon cancers that were negative for PTEN were excluded from the analysis). Data are represented as mean ± SEM. *P<0.05, Student's t-test.
Supplementary Figure 5. Co-knockdown of Akt and SGK3 inhibits the clonogenic potential of FHC normal colon epithelial cells.

(A) FHC cells transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) were transduced with the control shRNA (shControl), Akt shRNA (shAkt), SGK3 shRNA (shSGK3), or shAKT plus shSGK3. Twenty-four hours later, viable cells ($1 \times 10^4$) were subjected to clonogenic assays. The data shown are representative of three individual experiments. Scale bar, 1cm.

(B) Quantitation of colony formation as shown in Figure A. Data are represented as mean ± SEM of three individual experiments. **$P<0.01$, Student's $t$-test.
Supplementary Figure 6. A shRNA-resistant mutant form of INPP4B reverses the effects of INPP4B knockdown on PI(3,4,5)P₃, PI(3,4)P₂, and PI(3)P expression levels.

(A) WiDr and HCT116 cells stably transduced with shControl or shINPP4B1 were transduced with a shRNA-resistant mutant form of INPP4B (INPP4B-mut). Forty-eight hours later, the relative abundance of PI(3,4,5)P₃ was measured by using ELISA in lipid extractions of whole cells. Data are represented as mean ± SEM of three individual experiments. **P<0.01, Student's t-test.

(B) WiDr and HCT116 cells stably transduced with shControl or shINPP4B1 were transduced with a shRNA-resistant mutant form of INPP4B (INPP4B-mut). Forty-eight hours later, the relative abundance of PI(3,4)P₂ was measured by using ELISA in lipid extractions of cells. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.

(C) WiDr and HCT116 cells stably transduced with shControl or shINPP4B1 were transduced with a shRNA-resistant mutant form of INPP4B (INPP4B-mut). Forty-eight hours later, the relative abundance of PI(3)P was measured by using ELISA in lipid extractions of cells. Data are represented as mean ± SEM of three individual experiments. *P<0.05, Student's t-test.
Supplementary Figure 7. Overexpression of INPP4B increases PI(3,4,5)P_3, PI(3,4)P_2, and PI(3)P expression levels and reduces PTEN expression in colon cancer cells.
(A) The relative abundance of PI(3,4,5)P_3 in SW620 and HT-29 cells stably transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) was measured by using ELISA in lipid extractions of whole cells. Data are represented as mean ± SEM of three individual experiments.
*P<0.05, Student's t-test.
(B) The relative abundance of PI(3,4)P_2 in SW620 and HT-29 cells stably transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) was measured by using ELISA in lipid extractions of whole cells. Data are represented as mean ± SEM of three individual experiments.
*P<0.05, Student's t-test.
(C) The relative abundance of PI(3)P in SW620 and HT-29 cells stably transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) was measured by using ELISA in lipid extractions of whole cells. Data are represented as mean ± SEM of three individual experiments.
*P<0.05, Student's t-test.
(D) Whole cell lysates from SW620 and HCT116 cells stably transduced with the pCDH vector alone or INPP4B cDNA cloned into the pCDH vector (pCDH-INPP4B) were subjected to Western blot analysis of INPP4B, PTEN, and GAPDH (as a loading control). The data shown are representative of three individual experiments.
Supplementary Figure 8. Ets-1 transcriptionally regulates INPP4B in colon cancer cells.

(A) A schematic illustration of Ets-1 binding sites at the -279/-26 fragment of the INPP4B promoter.

(B) A schematic illustration of a luciferase reporter construct with or without the Ets-1 binding site-enriched fragment deleted for measurement of the transcriptional activity of the INPP4B promoter.

(C) SW620 and FHC cells transfected with the vector alone or Ets-1 cDNA were transiently transfected with the pGL3-basic-based reporter constructs (pGL3-vector, pGL3-INPP4B wt or pGL3-INPP4B mut, as shown in B). Twenty-four hours later, cells were subjected to the measurement of the luciferase activity. Data are represented as mean ± SEM of three individual experiments. **P<0.01, Student's t-test.
Supplementary Figure 9. Knockdown of Ets-1 reduces colon cancer cell proliferation, which is largely reversed by co-introduction of INPP4B.

WiDr and HCT116 cells transfected with the control siRNA or two Ets-1 siRNAs (Ets-1 siRNA1 and Ets-1 siRNA2) were transduced with the vector alone or INPP4B cDNA cloned in the pCDH vector (pCDH-INPP4B). Cells were subjected to BrdU incorporation assays. Data are represented as mean ± SEM of three individual experiments. *P<0.05, **P<0.01, Student's t-test.
Supplementary Figure 10. A Schematic illustration of the mechanism responsible for regulation of PI3K signalling by INPP4B in colon cancer cells.
In colon cancer cells, the protein phosphatase activity of INPP4B dephosphorylates PTEN and thus reduces its stability leading to its downregulation. This in turn results in increased PI(3,4,5)P$_3$, hydrolysis of which produces increased PI(3,4)P$_2$. Therefore, INPP4B promotes PI3K signalling through suppression of PTEN. This conceivably overrides the inhibitory effect of INPP4B on PI3K signalling through its hydrolyzing effect on PI(3,4)P$_2$. 
Supplementary Table 1. Summary of clinicopathological characteristics of the cohort of 124 colon cancer patients whose tissues were included in the IHC study

| Characteristics          | Cases | INPP4B expression levels in colon cancers (IRS) | P value<sup>2</sup> |
|--------------------------|-------|-----------------------------------------------|---------------------|
| Gender                   |       |                                               |                     |
| Male                     | 64    | 15.825 ± 5.132<sup>1</sup>                     | 0.6761              |
| Female                   | 60    | 15.300 ± 4.763                               |                     |
| Age at diagnosis         |       |                                               |                     |
| <59<sup>4</sup>          | 55    | 15.072 ± 4.193                               | 0.1971              |
| ≥59                      | 69    | 16.121 ± 5.566                               |                     |
| TNM Stage                |       |                                               |                     |
| I/II                     | 64    | 15.616 ± 5.072                               | 0.9176              |
| III/IV                   | 60    | 15.523 ± 4.856                               |                     |
| Histological Grade       |       |                                               |                     |
| I/II                     | 83    | 15.543 ± 4.688                               | 0.7401              |
| III                      | 41    | 15.888 ± 5.719                               |                     |

<sup>1</sup>IRS: Immunoreactive score
<sup>2</sup>Student's t-test; a P value less than 0.05 was considered statistically significant
<sup>3</sup>Data shown are mean ± SEM
<sup>4</sup>The median age of the patients in this cohort was 59
Supplementary Table 2. Summary of clinicopathological characteristics of the colon cancer patients whose tissues were included in the qPCR study

| Characteristics     | Cases | Relative abundance of INPP4B mRNA | P value |
|---------------------|-------|-----------------------------------|---------|
| Gender              |       |                                   |         |
| Male                | 69    | $8.126 \pm 3.586^1$               | 0.1103  |
| Female              | 51    | $9.258 \pm 3.931$                 |         |
| Age at diagnosis    |       |                                   |         |
| <60                 | 59    | $8.375 \pm 3.745$                 | 0.5254  |
| \geq 60             | 61    | $8.813 \pm 3.791$                 |         |
| TNM Stage           |       |                                   |         |
| I/II                | 62    | $8.550 \pm 3.642$                 | 0.8873  |
| III/IV              | 58    | $8.649 \pm 3.912$                 |         |
| Histological Grade |       |                                   |         |
| I/II                | 94    | $8.895 \pm 3.932$                 | 0.0648  |
| III                 | 26    | $7.574 \pm 2.968$                 |         |

1 Relative abundance of the expression was calculated as the level in laser capture micro-dissected CRC cells relative to the level in paired normal colon epithelial tissue that was arbitrarily designated as 1
2 Student’s t-test; a P value less than 0.05 was considered statistically significant
3 Data shown are mean ± SEM
4 The median age of the patients in this cohort was 60
Supplementary Table 3. Mutational status of KRAS, PIK3CA and PTEN in the colon cancer cell lines\(^1\)

| Cell line   | KRAS  | PIK3CA     | PTEN |
|-------------|-------|------------|------|
| SW480       | G12V  | W\(^2\)    | W    |
| SW620       | G12V  | W          | W    |
| HCT116      | G13D  | H1047R     | W    |
| EB          | G12D  | E545K      | W    |
| Lim1215     | W     | W          | W    |
| Lim1863     | W     | W          | W    |
| CaCO-2      | W     | W          | W    |
| WiDr        | W     | P449T      | W    |
| Colo205     | W     | W          | W    |
| HT-29       | W     | P449T      | W    |

\(^1\)Mutational status of the genes was determined by exon sequencing

\(^2\)W stands for wild-type
### Supplementary Table 4. The top 10 transcription factors that were differentially expressed in WiDr compared to SW620 cells

| Differences in expression | Genes   | Fold of changes$^2$ |
|---------------------------|---------|---------------------|
| Decreases                 | EGR1    | 62.3                |
|                           | SMAD9   | 54.1                |
|                           | GATA2   | 53.1                |
|                           | HNF1A   | 41.0                |
|                           | SMAD4   | 22.8                |
| Increases                 | ETS-1   | 11.6                |
|                           | RPLP0   | 4.3                 |
|                           | FOXA2   | 2.2                 |
|                           | ATF3    | 2.1                 |
|                           | PPARG   | 2.0                 |

$^1$The expression levels of the transcription factors were quantitated using a transcription factor qPCR array.

$^2$The relative abundance of a transcription factor in SW620 is arbitrarily designated as 1. The numbers stand for the expression levels in WiDr relative to SW620 cells.
Supplementary Materials and Methods

Cell culture and human tissues

Human colon cancer cell lines provided by Professor Gordon Burns (Faculty of Health and Medicine, University of Newcastle, Australia) and described previously were cultured in Dulbecco's Modified Eagle Medium (DMEM) (Sigma-Aldrich, Castle Hill, NSW, Australia) containing 5% FCS (Scientifix, Cheltenham, VIC, Australia).\(^1\) The normal human colon epithelial cell line FHC (ATCC\(^\text{®}\) CRL-1831\(^\text{TM}\)) was from ATCC, and cultured in a 1:1 mixture of Ham’s F12 and modified DMEM containing HEPES (25mM), cholera toxin (10 ng/ml), insulin (0.005 mg/ml), transferrin (0.005 mg/ml) and hydrocortisone (100 ng/ml) and 10% FCS. Individual cell line authentication was regularly confirmed every 6 months using the AmpFISTR Identifiler PCR Amplification Kit from Applied Biosystems (Mulgrave, VIC, Australia) and GeneMarker V1.91 software (SoftGenetics LLC, State College, PA, USA). Resulting cancer cell line STR profiles were cross-compared and, where available, matched with the ATCC’s online databases. Cell lines were tested for mycoplasma infection every month using Myco Alert according to the manufacturer’s protocol (Lonza, Walkersville, MD, USA). Human colon cancer tissues and paired noncancerous colon tissues were from patients undergoing surgical resection of sporadic colon cancers at the Department of Colorectal Surgery, Shanxi Cancer Hospital, China, and were prepared as described previously.\(^1\) TMA s were constructed from FFPE colon cancer and paired normal colon tissues retrieved from the Department of Pathology at Shanxi Cancer Hospital. Studies using human tissues were approved by the Human Research Ethics Committee of Shanxi Cancer Hospital, China. All participants provided written informed consent.

Antibodies and reagents

Antibodies against Akt (9272), phospho-Akt (Ser473) (4060), phospho-Akt (Thr308) (13038), phospho-SGK3 (Thr320) (5642), phospho-GSK3β (9323), GSK3β (12456), and phospho-PTEN (Ser380/Thr382/383) (9554) were from Cell Signalling Technology (Beverly, MA). Antibodies against INPP4B (sc-12318), SGK3 (sc-166847), Ets-1 (sc-55581), phospho-SGK1 (Ser422) (sc-16745) and PTEN (sc-7974) were purchased from Santa Cruz Biotechnology (Santa Cruz, CA). Antibody against p27 (610241) was purchased from BD Biosciences (San Jose, CA). Antibodies
against p21 (05-345), SGK1 (07-315) and phospho-PTEN (Ser385) (07-890) were from Millipore (Billerica, MA). Antibodies used for INPP4B (14543) and PTEN (9188) IP were from Cell Signalling Technology. Antibody used for INPP4B (ab81269) IHC was from Abcam (Cambridge, MA).

**Immunohistochemistry**

Immunohistochemical staining was performed on a Dako autostainer (Dako, Denmark). Antigen retrieval was performed in a pressure cooker for 30 s at 125 ºC. Antibody detection was performed using the Dako Envision HRP Detection system/DAB as per the manufacturer’s instructions. Slides were counterstained with haematoxylin. The specificity of antibodies was confirmed individually by pre-absorption with corresponding recombinant proteins, which eliminated immunoreactivity on tissue sections known to express the antigen. Quantification of immunostained cells was performed as described previously. In brief, the percentage of positive cells (0–100%) was estimated based on evaluation of immunohistochemically stained sections. Intensity of staining was judged on an arbitrary scale system of 0 - 4+: no staining (0), weakly positive staining (1+), moderately positive staining (2+), strongly positive staining (3+) and very strongly positive staining (4+). An immunoreactive score (IRS) was derived by multiplying the percentage of positive cells with staining intensity divided by 10. Pathologists were blinded to the group allocation when quantitating immunostaining. When comparing PTEN expression levels between colon cancers with high and low levels of INPP4B expression, the median of INPP4B levels were defined as the cutoff point (center values).

**Colon cancer xenograft mouse model**

Five- to six-week-old male athymic nude BALB/c mice were purchased from Model Animal Research Centre of Nanjing University, China. The mice were raised in the Animal Centre of Shanxi Cancer Hospital and Institute according to the regulation of the Animal Research Ethics Committee of Shanxi Cancer Hospital, China. HCT116 cells with or without INPP4B stably knocked down co-introduced with myr-Akt or myr-SGK3 were injected subcutaneously into each flank of the mice. Tumor growth was monitored with a sliding caliper three times per week. Mice were sacrificed and tumors were weighed at 36 days after transplantation. No randomization or blinding was involved. Studies on animals were approved by the Animal Research Ethics Committee of Shanxi Cancer Hospital, China.
**Immunoblotting**

Immunoblotting was carried out as described previously.\(^5\)

**Cell viability**

Cell viability was quantitated using the CellTiter-Glo Luminescent Cell Viability Assay kit (Promega, San Luis Obispo, CA) as described previously.\(^6\) Luminescence was recorded by Synergy 2 multidetection microplate reader (BioTek, VT).

**BrdU proliferation assays**

BrdU cell proliferation assays were carried out using the BrdU Cell Proliferation Assay kit (Cell Signaling) as described before.\(^5\) Briefly, cells were seeded at 5x10\(^3\) cells per well in 96-well plates overnight before treatment as desired. BrdU (10 µM) was added and cells were incubated for 4 hours before BrdU assays were carried out. Absorbance was read at 450 nm using a Synergy 2 multidetection microplate reader (BioTek, VT).

**Clonogenic assays**

Clonogenic assays were carried out as described previously.\(^5\) In brief, cells were seeded at 2000 cells/well onto 6-well culture plates. Cells were then allowed to grow for a further 12 days before fixation with methanol and staining with crystal violet (0.5% solution).

**Anchorage-independent cell growth**

5x10\(^4\) FHC cells transduced with vector alone or INPP4B cDNA were seeded in 0.3% cell agar layer, which was on top of 0.6% base agar layer in 12-well culture plates. Cells were then incubated for a further 30 days at 37 °C and 5% CO\(_2\). Cell colony formation was then examined under a light microscope.

**Immunoprecipitation**
Immunoprecipitation was carried out as described previously. Briefly, 400 µl of whole cell lysate were precleared by incubation with 50 µl of protein A/G beads (Thermo Fisher Scientific, Scoresby, VIC, Australia) on a rotator at 4 °C for 1 hour. 5µg of antibody or corresponding control IgG were prepacked with 50 µl of protein A/G beads on a rotator at 4 °C for 1 hour. The precleared lysate were added into the prepacked beads and incubated on a rotator at 4 °C overnight. The beads were then pelleted by centrifugation and washed three times with ice-cold PBS-Tween. Proteins were then eluted with 50 µl of Elution buffer (Thermo Fisher Scientific) and neutralized by Neutralization Buffer (Thermo Fisher Scientific). Eluted protein was used for PTEN lipid phosphatase assay, protein phosphatase assay, and western blotting.

**Short hairpin RNA (shRNA)**

shRNA was carried out as described before. In brief, MISSION® human shRNA lentiviral transduction particles: INPP4B (TRCN0000230837 and TRCN0000230838), SGK3 (TRCN0000199867) as well as the corresponding control particles were purchased from Sigma-Aldrich. The human Akt shRNA kit (TG320629) and scramble shRNA kit (TG300130), in which shRNA constructs are in retroviral GFP vector (pGFP-VRS), were purchased from Origene (Australian Biosearch, Karrinyup, WA, Australia). These shRNAs were used to infect cells according to the manufacturer’s protocol.

**Quantitative reverse transcription-PCR (qPCR)**

qPCR was performed as described previously. The primer sequences are: INPP4B, forward, 5’-CCC CGG GTA CTG AGG CTT CG-3’, reverse, 5’-CTT TGT ATT CTC TCC CGG AGG CG-3’; Ets-1, forward, 5’ GTC GTG GTA AAC TCG G-3’, reverse, 5’-CAG CAG GAA TGA CAG G-3’. The relative expression level of INPP4B or Ets-1 mRNA was normalized against β-actin mRNA.

**Luciferase reporter assays**

The -279/-26 fragment of the *INPP4B* promoter was cloned by PCR using human genomic DNA as a template. The fragment was then cloned into promoter-less luciferase reporter plasmid pGL3-Basic
Luciferase Vector (Promega). Cells were transiently transfected with pGL3-vector or pGL3-INPP4B as well as pRL-TK Renilla luciferase control vector with or without co-transfection desired constructs. Luciferase activity was measured using a Synergy 2 multi-detection microplate reader (BioTek, VT).
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