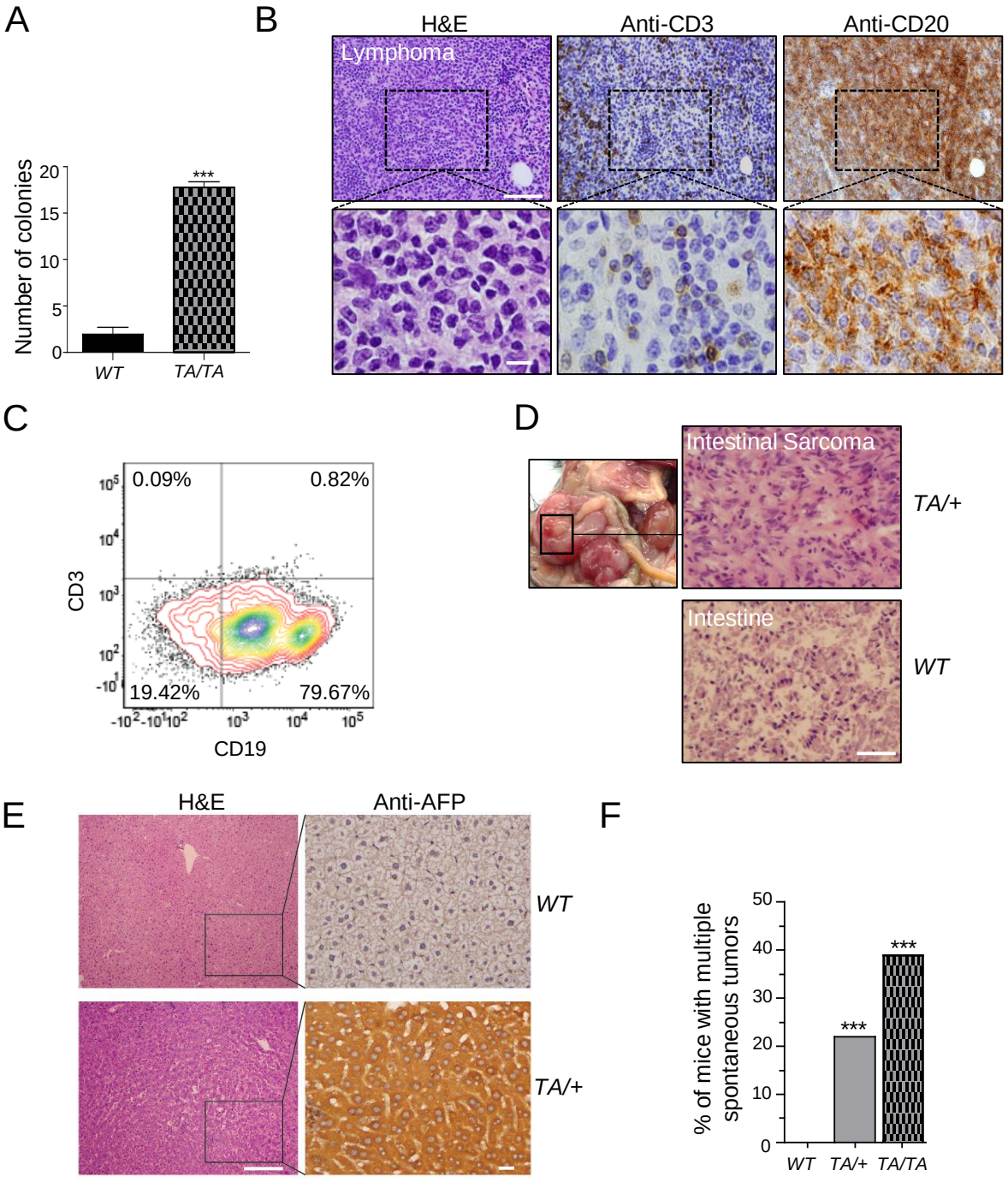
Supplementary Information

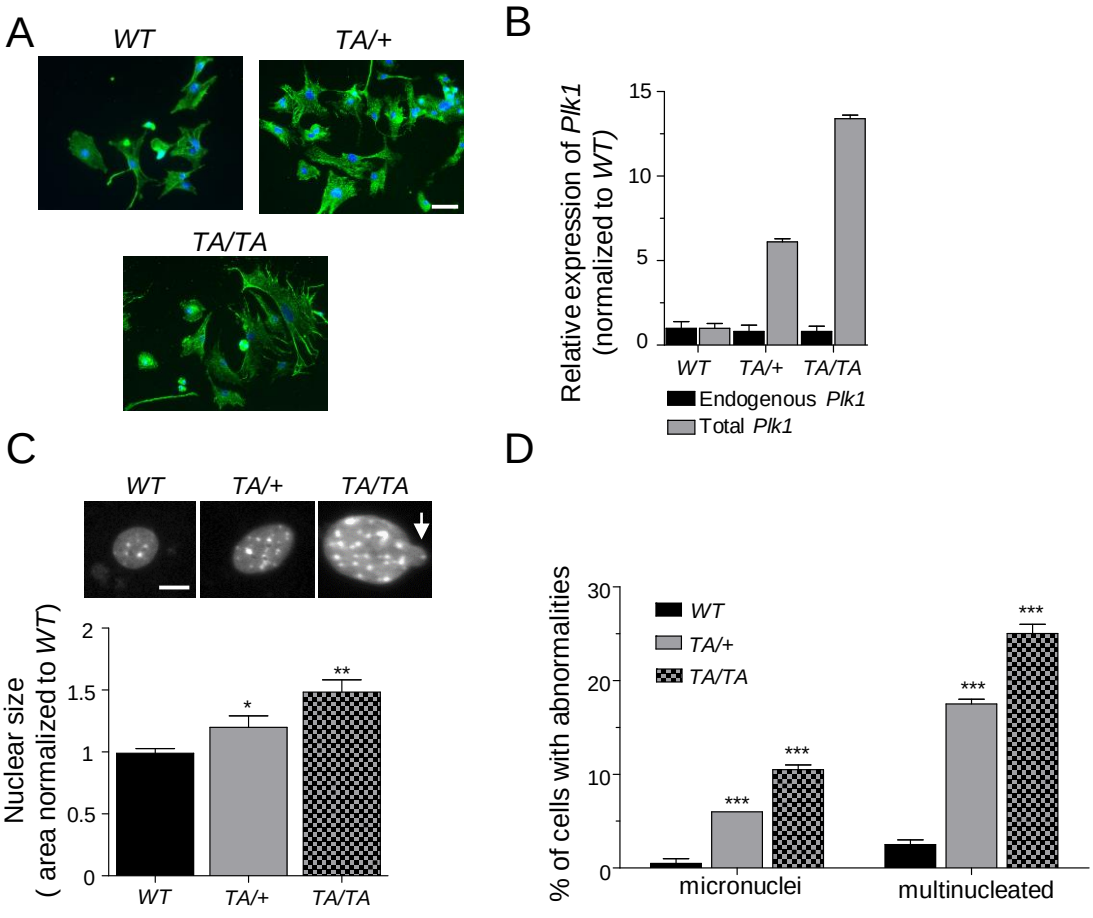
Supplementary Figures

Figure S1


A
Number of colonies
20
15
10
5
0
***
WT
TA/TA
B
H&E
Anti-CD3
Anti-CD20
Lymphoma
C
0.09%
0.82%
19.42%
79.67%
CD3
CD19
D
Intestinal Sarcoma
TA/+
Intestine
WT
E
H&E
Anti-AFP
WT
TA/+
F
% of mice with multiple spontaneous tumors
50
40
30
20
10
0
***
***
WT
TA/+
TA/TA

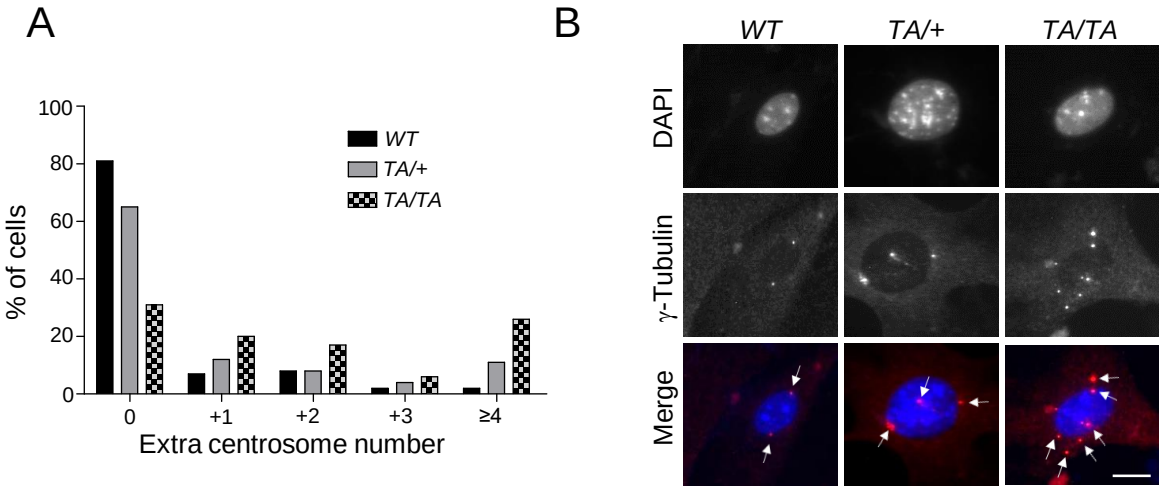
Supplementary Fig. S1: *Plk1* overexpression induces transformation and spontaneous tumor development. (Related to Fig. 2). **(A)** Spontaneous immortalized *WT* and *Plk1*$^{TA/TA}$ transgenic MEFs were plated in soft agar and grown for 21 days. Graph represents the number of colonies counted for each genotype. The experiment was repeated 3 times. (Mean ± SEM, ***: $p < 0.001$ Student's *t* test). **(B)** Immunohistochemistry (IHC) staining of CD3 and CD20 proteins of representative spontaneous lymphoma derived from *Plk1* transgenic mice with corresponding H&E staining. Top **s**cale bar 100µm, bottom scale bar 20µm. **(C)** CD3 and CD19 cell surface expression was assessed by flow cytometry in cells from a representative spontaneous lymphoma collected from a *Plk1* transgenic mouse. Numbers in each quadrant indicate percentage of the total population. **(D)** A representative spontaneous intestinal sarcoma from transgenic *TA/+* mice along with its corresponding H&E staining. Staining of *WT* intestine tissue is shown below. Scale bar: 100 µm. **(E)** IHC staining of AFP protein in liver tissues from *WT* and *Plk1* transgenic mice with corresponding H&E staining. Left scale bar 100 µm; right scale bar 20 µm. **(F)** Incidence of *WT* and *Plk1* transgenic mice with multiple spontaneous tumors. (*WT* vs *TA/+* or *WT* vs *TA/TA*: ***: $p < 0.001$, Chi-squared test).

Figure S2



Supplementary Fig. S2: *Plk1* overexpression induces aneuploidy in PAEpiCs derived from *Plk1* transgenic mice. (Related to Fig. 3). **(A)** Representative images of *WT*, *TA/+* and *TA/TA* PAEpiCs stained with DAPI (blue) and anti-EpCAM, a marker of epithelial cells (green). Scale bar: 20 µm. **(B)** qRT-PCR for total (endogenous and exogenous) and endogenous *Plk1* transcript in PAEpiC cells of indicated genotypes**. (C**) (Top) Representative images of *WT*, *TA/+*, and *TA/TA* PAEpiC stained with DAPI. (Bottom) Graph representing nuclear size of *WT*, *TA/+*, and *TA/TA* PAEpiCs, normalized to *WT* PAEpiCs. Fifty cells/genotype, repeated 3x. (Mean ± SEM, *: $p < 0.05$; **: $p < 0.01$). Scale bar: 10 µm. Arrow: nuclear buds**. (D**) Graph representing percentage of micronucleated or multinucleated *WT*, *TA/+*, and *TA/TA* PAEpiCs. Fifty cells/genotype, repeated 3x. (Mean ± SEM, ***: $p < 0.001$). (**E**) Graph representing percentage of *WT*, *TA/+*, and *TA/TA* PAEpiCs with gains of whole chromosome(s). One hundred cells/genotype, repeated 2x. (**: $p < 0.01$, Chi-squared test).

Figure S3



Supplementary Fig. S3: *Plk1* overexpression leads to centrosome amplification in *Plk1* transgenic PAEpiCs. (Related to Fig. 4). **(A)** Centrosome count of *WT*, *TA/+*, and *TA/TA* PAEpiCs immunostained for γ-Tubulin. One-hundred metaphases/genotype counted. **(B)** Representative images of *WT* and *Plk1* transgenic PAEpiC cells immunostained for γ-Tubulin (red) and DAPI (blue). Scale bar: 10 µm. White arrow: centrosome.

Figure S4

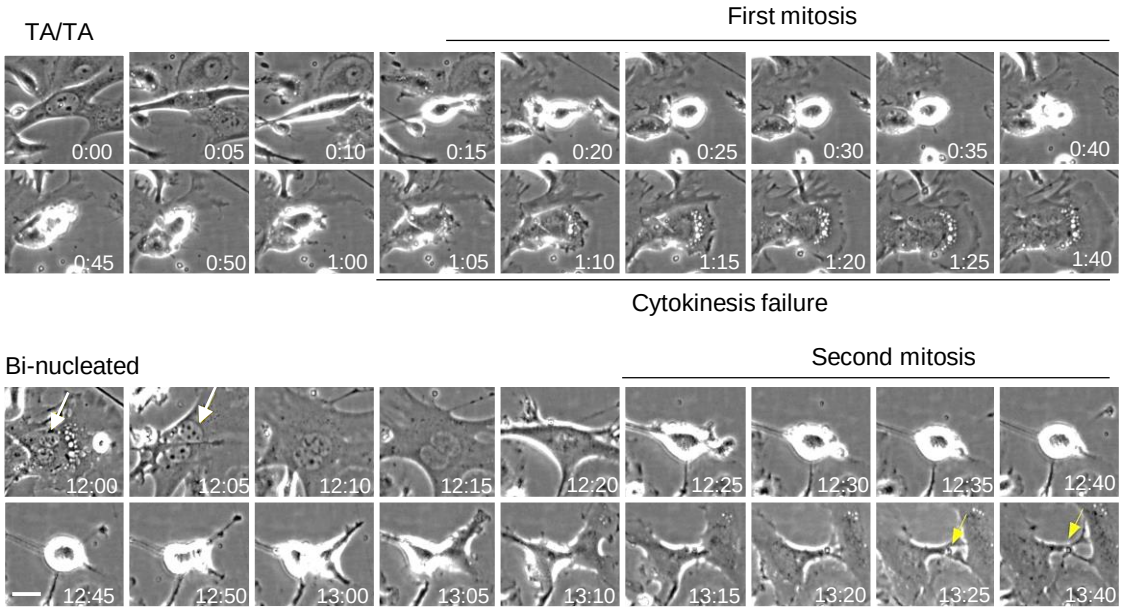
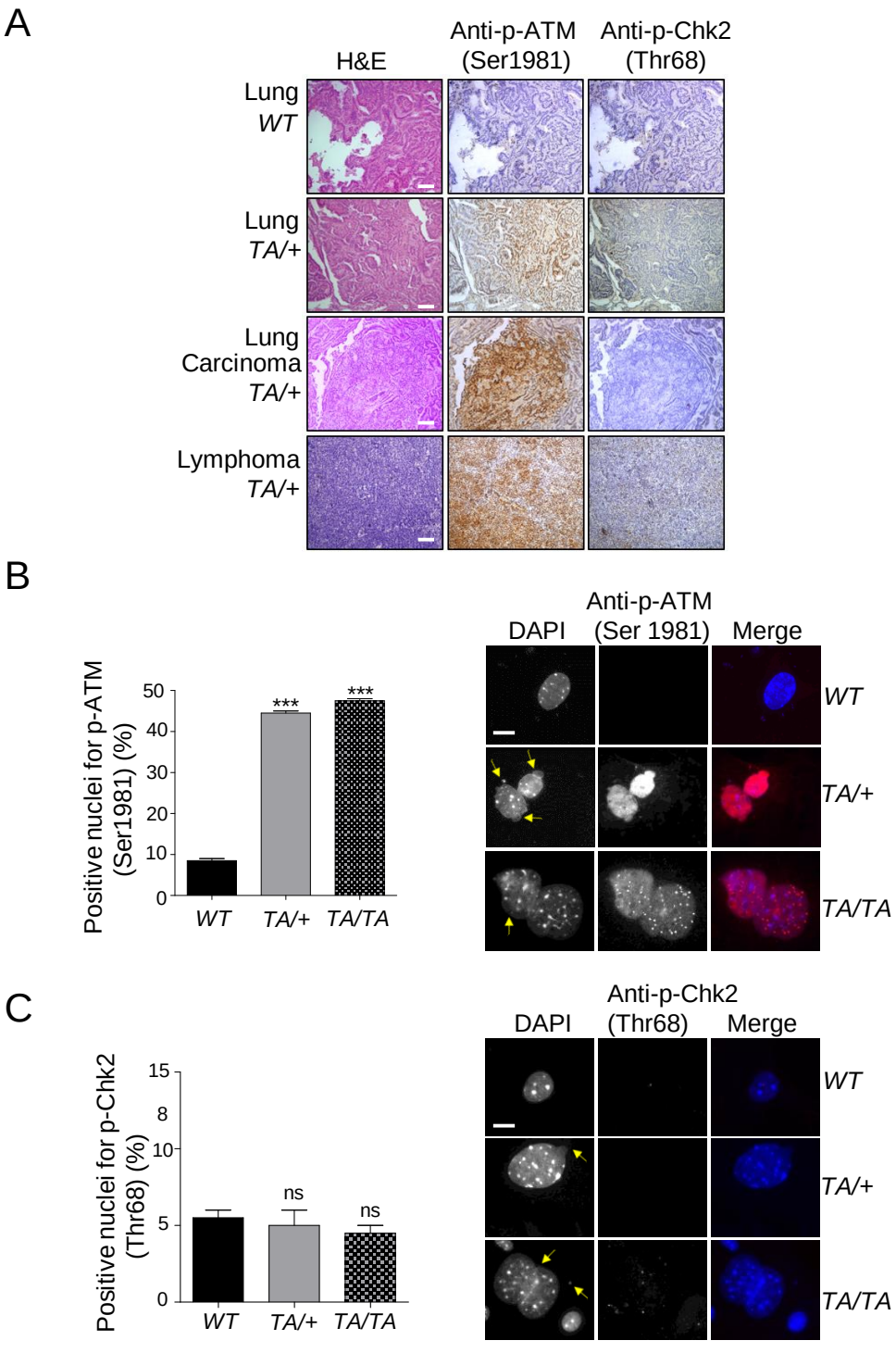


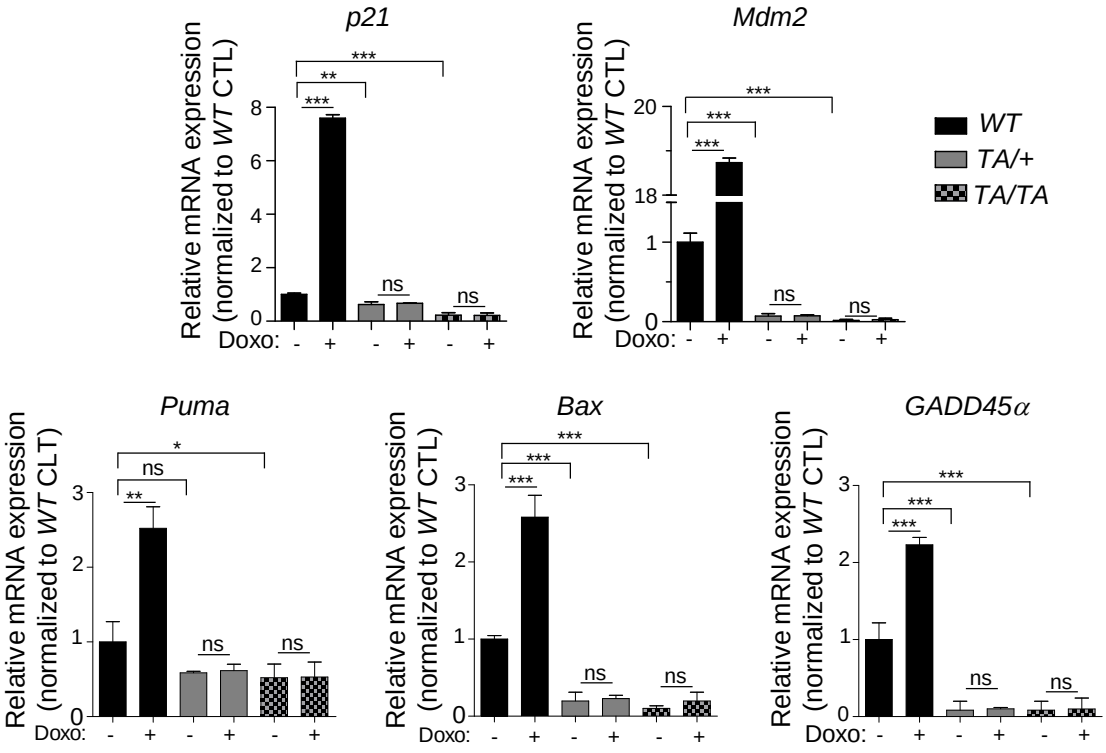
Figure S4: *Plk1* overexpression promotes proliferation of giant polyploidy PAEpiC. Representative binucleated *TA/TA* PAEpiC monitored during 2 cell divisions. Arrow: -White : binucleated cell. – Yellow: abscission failure. Scale bar: 10 mm.

Figure S5


A
H&E
Anti-p-ATM (Ser1981)
Anti-p-Chk2 (Thr68)
Lung WT
Lung TA/+
Lung Carcinoma TA/+
Lymphoma TA/+
B
Positive nuclei for p-ATM (Ser1981) (%)
50
40
30
20
10
0
***
***
WT
TA/+
TA/TA
DAPI
Anti-p-ATM (Ser 1981)
Merge
WT
TA/+
TA/TA
C
Positive nuclei for p-Chk2 (Thr68) (%)
15
8
10
5
0
ns
ns
WT
TA/+
TA/TA
DAPI
Anti-p-Chk2 (Thr68)
Merge
WT
TA/+
TA/TA

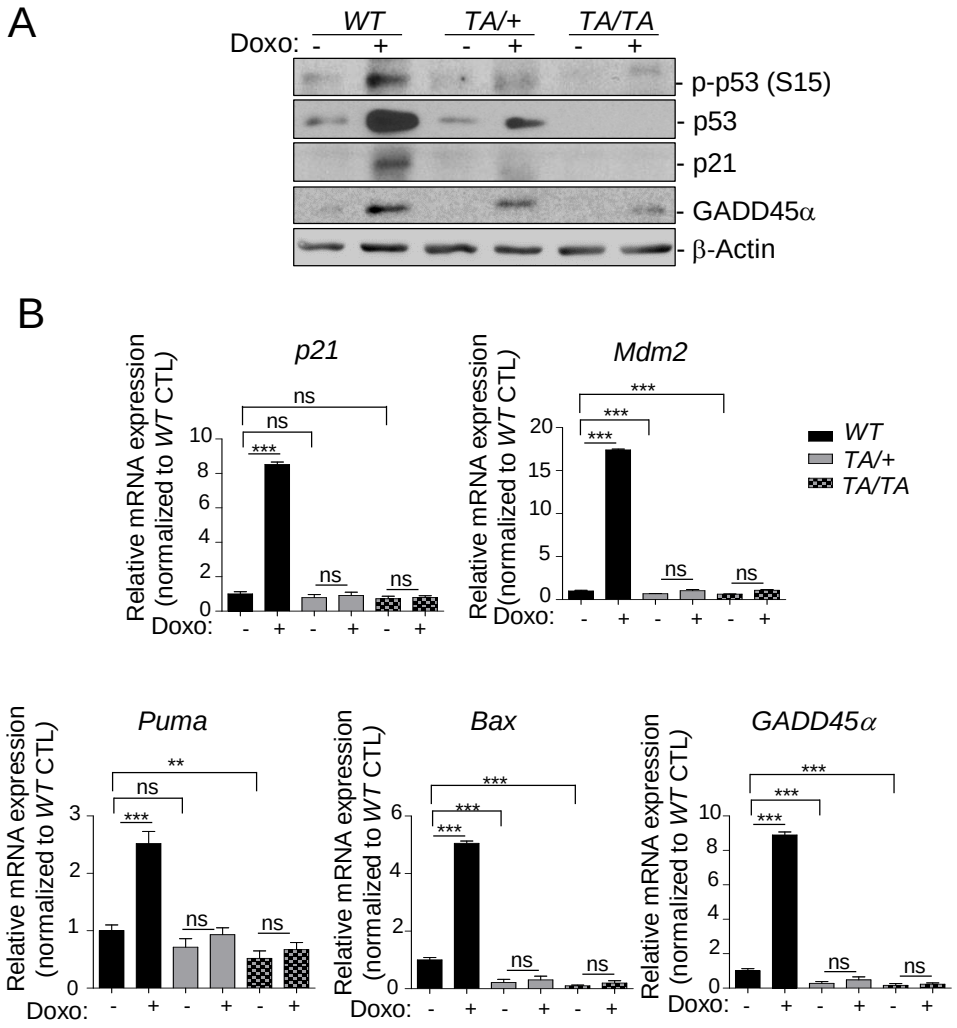
Supplementary Fig. S5: The effect of *Plk1* overexpression on the ATM/Chk2 signaling. (Related to Fig. 6). **(A)** IHC staining of phospho-ATM (Ser1981) and phospho-Chk2 (Thr68) in nonmalignant and malignant (carcinoma and lymphoma) tissues from *Plk1*$^{TA/+}$ transgenic mice, as well as lung tissues from *WT* mice, along with corresponding H&E staining. Scale bar: 100 µm. **(B-C)** (Left) Percentage of PAEpiCs of indicated genotypes positive for phospho-ATM (Ser1981) (B) and phospho-Chk2 (Thr68) (C) staining. 100 cells/genotype, repeated 2x. (Mean ± SEM, *WT* vs *TA/+* or *TA/TA*: ***: $p < 0.001$). (Right) Representative images from PAEpiCs of indicated genotypes immunostained for phospho-ATM (Ser1981) (B) and phospho-Chk2 (Thr68) (C) in red and Dapi in blue. Scale, 10 µm. Yellow arrow: nuclear abnormalities (micronuclei, bi-nucleated cells, and nuclear buds).

Figure S6



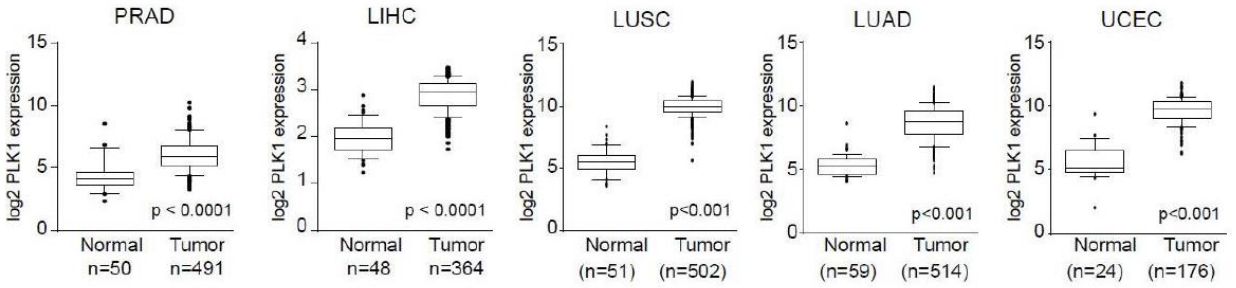
Supplementary Fig. S6: The expression of p53 target genes in *Plk1* transgenic PMEFs. (Related to Fig. 6). qRT-PCR analysis of 5 canonical targets of p53 (*p21*, *Mdm2*, *Puma*, *Bax*, and *GADD45α*) in PMEFs with the indicated genotypes in the presence or absence of doxo (0.5 mM for 6 hrs). GAPDH was used as internal control. Repeated 3x. (Mean ± SEM, *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$, t-test).

Figure S7



Supplementary Fig. S7: *Plk1* overexpression compromises the DNA-damage response in PAEpiCs. (Related to Fig. 6)**. (A)** PAEpiCs of the indicated genotypes were treated with or without Doxo (0.5 mM for 2 hrs). p53 expression and activity (phosphor-Ser15), and its target gene expression (*p21* and *GADD45α*) were examined by Western blotting. (**B**) qRT-PCR analysis of 5 canonical targets of p53 (*p21*, *Mdm2*, *Puma*, *Bax*, and *GADD45α*) in PAEpiCs of the indicated genotypes in the presence or absence of doxo (0.5 mM for 6 hrs)**.** GAPDH was used as internal control. Repeated 2x. (Mean ± SEM, *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$ t-test)

Figure S8



Supplementary Fig .S8 : *PLK1* is overexpressed in human cancers. (Related to Fig. 7) Comparison of PLK1 expression levels in PRAD, LIHC, LUSC, LUAD, UCEC cohorts and their corresponding normal tissues.

Supplementary Video Legends

Video 1: *Plk1* overexpression promotes lagging chromosomes and the formation of micronucleated cells. *TA/+* PMEFs expressing H2B-mCherry were recorded (3 min/image) as cells entered mitosis. White arrows indicate micronuclei.

Video 2: *Plk1* overexpression promotes lagging chromosome and cytokinesis failure, resulting in formation of binucleated progeny. *TA/TA* PMEFs expressing H2B-mCherry were recorded (every 3 min/image) as cells entered mitosis. White arrow indicates chromatin bridges.

Video 3: *Plk1*-overexpressing PMEFs display an increase in centrosomes number, leading to multipolar spindles and mitotic errors. *WT* (Left) and *TA/TA* PMEFs (Right) co-expressing H2BmCherry and EGFP-α-Tubulin were recorded (every 3 min/image) as cells entered mitosis. White arrows indicate centrosomes.

Video 4: *Plk1* overexpression promotes proliferation of cells with CIN. Video 4A: A binucleated *TA/TA* PMEF expressing H2B-mCherry was recorded (every 3 min/image) as the cell entered mitosis. Chromosome segregation failure led to the formation of progeny cells with micronuclei. **Video 4B:** A binucleated *TA/TA* PMEF expressing H2B-mCherry was recorded (every 3 min/image) as the cell entered mitosis. The failures in chromosome segregation and cytokinesis led to the formation of a giant multinucleated progeny cell.

Video 5: Multinucleated *Plk1* PMEFs are able to progress through a second round of cell division. *TA/TA* PMEFs were recorded by phase-contrast video-microscopy for 24 hrs at a rate of 4 min/image. Red arrow points out a binucleated cell that progressed through 2 rounds of cell division with cytokinesis failure, leading to the formation of a giant multinucleated progeny cell.

Supplementary Materials and Methods

Antibodies

Antibodies	SOURCE	IDENTIFIER
anti-GFP	GeneTex	GTX113617
anti-PLK1	Millipore	clone 35-206
anti-phospho-Histone H3 (Ser10)	Millipore	06-570
anti-γ Tubulin	Santa Cruz	sc-17787
Pacific Blue anti-Annexin V	BioLegend	640917
PE anti-CD3	BioLegend	100205
FITC anti-CD19	BioLegend	152403
anti-CD326 (Ep-CAM)	BioLegend	clone G8.8
anti-CD31	BioLegend	MEC13.3
anti-CD45	BioLegend	30-F11
c-PARP-1	Cell signaling	Asp214
c-caspase 3	Cell signaling	Asp175
anti-p53 (A-1)	Santa Cruz	sc-393031
Phospho-p53 (Ser15)	Cell signaling	#9284
Anti-p21	Santa Cruz	sc-6246
anti-BubR1	Abcam	ab54894
anti-Cdc20	cell signaling	14866
Anti-rabbit polyclonal AFP	Proteintech	14550-1-AP
anti-phospho-ATM Ser1987	Abcam	ab81292
phospho-Chk2 Thr68	GeneTex	GTX55055
anti-Ki67	Abcam	ab15580
β-Actin	Santa Cruz	sc-47778
anti-α-Tubulin	Sigma	#T5168
anti-GAPDH	Santa Cruz	sc-32233
anti-rabbit IgG-HRP	Santa Cruz	sc-2357
anti-mouse IgG-HRP	Santa Cruz	sc-516102

Primers for mouse genotyping

Primers for genotyping	
Forward primer for Hprt-Cre:	5'-ACC AGC CAG CTA TCA ACT CG -3'
Reverse primer for Hprt-Cre:	5'-TTACATTGGTCCAGCCACC -3'
Forward primer for NEO:	5'-AGGATCTCCTGTCATCTCACCTTGCTCCTG-3'
Reverse primer for NEO:	5'-AAGAACTCGTCAAGAAGGCGATAGAAGGCG-3'
Forward primer for PLK1:	5'-GGCGGCTCTAGAGCCTCTGCTAACC-3'
Reverse primer for PLK1:	5'-CTCGAAGCATTTGGCGAAGCCTCC-3'

Isolation and culture of Pulmonary alveolar epithelial cells (PAEpiCs)

PAEpiCs were isolated from 2-month-old control and *Plk1* transgenic mice according a previously described procedure (1). Briefly, mice were euthanized using an approved inhaled procedure. Cardiac perfusion with 10-mL D-PBS from the right ventricle was performed. The trachea was exposed and intubated with a 20-gauge catheter, allowing perfusion of the lungs with 2-mL dispase. After infiltration with dispase, the lungs were isolated from the thoracic cavity and

incubated for 20 min in 5-mL dispase at room temperature on an agitation table. The lungs were then minced using scissors and transferred in 10-mL complete DMEM containing 10 mM HEPES, 10% FBS, 1% penicillin-streptomycin, and 1% non-essential amino acids. After mincing, 100 µL of DNase I (1 mg/mL) was added to the sample and incubated for 10 min at room temperature on an agitation table. The cells were sequentially filtered through 70-µm and 40-µm filters, and 500 µL of AKC lysis buffer was added to remove the red blood cells. The cell suspension was then centrifuged for 10 min at 1500 rpm at room temperature. The cell pellet was resuspended in 500-µL complete DMEM for selection of PAEpiCs using the magnetic bead selection procedure. The cell suspension was first incubated with anti-CD45, anti-CD16/32, and anti-CD31 conjugated dynabeads for 1 hr to remove hematopoietic cells (CD45 positive), alveolar macrophages (CD45 positive and CD16/32 positive), and endothelial cells (CD31 positive) from the suspension. The suspension was then incubated with anti-EpCAM (epithelial marker)-conjugated dynabeads for 1 hr at 37°C. A magnetic separator was used to sort the bead-bound cells ($EpCAM^+$). Isolated cells were resuspended in complete DMEM and plated in an appropriately sized culture flask. The purity of PAEpiCs was assessed by immunostaining using anti-EpCAM antibody (supplemental Fig. S2A). PAEpiCs were maintained in DMEM (containing 10% fetal bovine serum [FBS], 1% penicillin-streptomycin, and 1% non-essential amino acids, 10mM HEPES).

Metaphase spread

Chromosome counts were performed on the metaphase spreads of P1 PMEFs, PAEpiCs, and lymphomas from *Plk1* transgenic mice. Cells were treated with 0.5-µg/mL colcemid for 6 h, and collected by centrifugation. The cells were incubated in a hypotonic solution (0.075-M KCl) for 30 min at 37°C and 5 drops of fixation solution (methanol/acetic acid 3:1) were added before centrifugation at 1200 rpm for 10 min. Supernatant was removed and cells were resuspended in

fixation solution and incubated at 4℃ for 10 min, followed by rinsing 3 times with fixation solution. The cells were dropped onto glass slides in a humidified chamber (45% humidity, 22℃). The slides were dried and stained in trypsin-Giemsa solution. The stained slides were examined under a light microscope using Cytovision software. Chromosomes from 100 cells per genotype were counted.

Quantitative real-time PCR

Total RNA was extracted from cells or tissue using Trizol (Thermo Fisher Scientific the RNeasy Mini Kit (Qiagen) and cDNAs were generated with the Script Reverse Transcription Supermix Kit (Bio-Rad), according to the manufacturer's instructions. qRT-PCR reactions were performed using a StepOne Plus (Applied biosystem) thermal cycler and the SYBR Green Mix (Thermo Scientific). Data were evaluated by the ΔΔCt method with GAPDH as a housekeeping gene for normalization. Primer sequences:

Primers for qRT-PCR	
Trans-PLK1-Forward (p1):	5'-AATTCCGGAGGTCCTAGTGG-3'
Trans-PLK1-Reverse (p2):	5'-CTACGACGTGTTGGTGTGCT-3'
Endo-PLK1-Forward (p3):	5'-CAGGTTCGTGGAGCAACTT-3'
Endo-PLK1-Reverse (p4):	5'-GGACCTCCGGAATTTCTTTC-3'
p21-Forward:	5'-CAAGAGGCCCAGTACTTCCT-3'
p21-Reverse:	5'-ACACCAGAGTGCAAGACAGC-3'
Bax-Forward:	5'-TAGCAAACTGGTGCTCAAGG-3'
Bax-Reverse:	5'-TCTTGGATCCAGACAAGCAG-3'
Mdm2-Forward:	5'-CTAGCTTCTCCCTGAATGCC-3'
Mdm2-Reverse:	5'-TTGCACACGTGAAACATGAC-3'
Puma-Forward:	5'-GTACGAGCGGCGGAGACAAG-3'
Puma-Reverse:	5'-GCACCTAGTTGGGCTCCATTTCTG-3'
GADD45α-Forward:	5'-GCGGTTCAGAAGATGCAGGC-3'
GADD45α-Reverse:	5'-GGTTGTGCCCAATGTCTCCG-3'
GAPDH-Forward:	5'-TGCACCACACCAACTGCTTAGC-3'
GAPDH-Reverse:	5'-TGGATGCAGGGATGATGTTC-3'

Analysis of p53 mutations by Sanger sequencing

To identify potential mutations in the *p53* gene, genomic DNA was extracted from PMEFs, immortalized MEFs, lungs, spleens, and derived tumors of *WT*, *Plk1*$^{TA/+}$, and *Plk1*$^{TA/TA}$ mice using

QIAamp DNA Mini Kit in accordance with the manufacturer's instructions. The coding regions of the *p53* gene were amplified by PCR. Multiple primer sets were used covering all coding regions of *p53* (exons 2-11) as described in (2). These primers sets were designed using a serial linked pipeline to ensure high fidelity of the sequencing results. PCR primers for amplifying genomic DNA sequences within exons 2-11 of *p53* gene are summarized below. The PCR mixture (50 µL) consisted of 200 ng of DNA template, 1.5 µL of each forward and reverse primer (10 µM), 1µL of high-fidelity Hot Start KOD DNA polymerase, 3 µL of $MgCl_2$ (25 mM), 5 µL PCR buffer, and 5µL of dNTP mix (2mM each). PCR amplification was programmed with an initial denaturation step at 95°C for 5 min, followed by 35 cycles of denaturation, annealing and primer extension. The annealing cycle was set to 60°C for 30 s per cycle. The conditions for the primer extension step were 70°C for 30 s per cycle. Upon completion of the cycling steps, a final extension step was performed at 70°C for 5 min. The PCR products were then purified using QIAquick Gel Extraction Kit (QIAGEN) in accordance with the manufacturer's instructions. The PCR products were analyzed by Sanger sequencing, which was performed by Eurofins Genomics. Sequences from *WT*, *Plk1*$^{TA/+}$, and *Plk1*$^{TA/TA}$ tissue were aligned and compared among them, and to the mouse reference genome of *p53* (NCBI) using the Multiple Sequence Alignment website Clustal Omega. Primer sequences:

Primers for p53 sequencing	
p53-E2-Forward	5'- CTCGTGTAAAACGACGGCCAGTCCCTACTGGATGTCCCACCTTC-3
p53-E2-Reverse	5'-CTGCTCAGGAAACAGCTATGACTTTGTTTCTCTCAGGCAAGGG-3'
p53-E3-Forward	5'-CTCGTGTAAAACGACGGCCAGTTTCAAACATGGTATGGTGTTGGG-3'
p53-E3-Reverse	5'-CTGCTCAGGAAACAGCTATGACTGGATGGGACAAAGAAGAACCC-3'
p53-E4-Forward	5'-CTCGTGTAAAACGACGGCCAGTGTTGGGCTGGTAGGCTGAGAAC-3'
p53-E4-Reverse	5'-CTGCTCAGGAAACAGCTATGACAGAGGCATTGAAAGGTCACACG-3'
p53-E5-Forward	5'-CTCGTGTAAAACGACGGCCAGTCACCTGATCGTTACTCGG5'-CTTG
p53-E5-Reverse	5'-CTGCTCAGGAAACAGCTATGACTGCCTGTCTTCCAGATACTCGG-3'
p53-E6-Forward	5'-CTCGTGTAAAACGACGGCCAGTCTGCTCCGATGGTGATGGTAAG-3'
p53-E6-Reverse	5'-CTGCTCAGGAAACAGCTATGACACTCAGCGTCTCTATTTCCCGC-3'
p53-E7-Forward	5'-CTCGTGTAAAACGACGGCCAGTAGGTAGGGAGCGACTTCACCTG-3'
p53-E7-Reverse	5'-CTGCTCAGGAAACAGCTATGACCTCGTGGAACAGAAACAGGCAG-3'
p53-E8-Forward	5'-CTCGTGTAAAACGACGGCCAGTTCTTACTGCCTTGTGCTGGTCC-3'

p53-E8-Reverse	5'-CTGCTCAGGAAACAGCTATGACAATGGGTACAGCTTGTCTCTGGC-3'
p53-E9-Forward	5'-CTCGTGTAAAACGACGGCCAGTGGAAGTCCTTTGCCCTGAACTG-3'
p53-E9-Reverse	5'-CTGCTCAGGAAACAGCTATGACCTGAGAACCACTGTCGGAGGAG-3'
p53-E10-Forward	5'-CTCGTGTAAAACGACGGCCAGTTCGTGAAAGTGGTTGTGTGACC-3'
p53-E10-Reverse	5'-CTGCTCAGGAAACAGCTATGACTGCAGCCCTAAGCATCTAGCAG-3'
p53-E11start-Forward	5'-CTCGTGTAAAACGACGGCCAGTTCCCTACCCATAGTAGAAGCCATC-3'
p53-E11start-Reverse	5'-CTGCTCAGGAAACAGCTATGACGGGATGCAGAGGCAGTCAGTC-3'
p53-E11end-Forward	5'-CTCGTGTAAAACGACGGCCAGTAGCTCCCATCACTTCATCCCTC-3'
p53-E11end-Reverse	5'-CTGCTCAGGAAACAGCTATGACGGCCAGCAGAGACCTGACAAC-3'

Fluorescence in situ hybridization

Paraffin sections were analyzed by FISH. Probes from 2 mouse bacterial artificial chromosome (BAC) clones (Empire Genomic) of chromosome 6 (RP23-20F16) and chromosome 18 (RP23-263F6) were labeled with Spectrum Red-dUTP and Spectrum Green-dUTP, respectively. The number of hybridization signals for these probes was assessed in 100 interphase nuclei with well-delineated contours.

Lentivirus preparation and infection

Lentiviral vectors pLenti6-H2B-mCherry (addgene Plasmid #89766), and L304-EGFP-Tubulin-WT (addgene, Plasmid #64060) along with packing (psPAX2) and envelope (pMD2.G) vectors were transfected into 293T cells using PEI. The supernatants were collected after transfection for 48 hrs and 72 hrs, filtered through a 0.45 μm filter, and concentrated by ultracentrifugation at 28,000 rpm at 4℃ for 2 hrs. Resuspended viral pellets in the presence of 8 μg/mL of polybrene were used to infect cells.

Senescence-associated β-galactosidase activity

Cells were fixed with 2% paraformaldehyde and 0.2% glutharaldehyde in PBS, pH 7.4, for 5 min and incubated at 37°C overnight in staining solution (40-mM sodium citrate, pH 6.0, 1% X-

gal, 5-mM potassium ferrocyanide, 5-mM ferricyanide, 150-mM NaCl, and 2-mM $MgCl_2$). Cultures were examined under phase-contrast microscopy.

Subcellular protein fractionation

Cells were lysed in hypotonic buffer (10-mM HEPES-KOH, 1.5-mM $MgCl_2$, 10-mM KCl, 0.5-mM DTT, 0.2-mM PEFA 1023, pH 7.9, 0.5% NP-40). Cell lysates were centrifuged for 10 s at 16 000 g at 4°C. The supernatants were collected (cytoplasmic extracts), and the pellets were washed twice with hypotonic buffer and lysed with high-salt buffer (450-mM NaCl, 1-mM PMSF, 50-mM Tris pH 7.4, 0.2-mM Na_3VO_4, 5-mM β-glycerophosphate, 20% glycerol, 2-mM DTT, 1% NP-40), following incubation for 10 min at 4°C (nuclear extracts).

Meta-analysis

Genomic segmentation profiling and PLK1 expression levels from a large collection of LIHC, PRAD, SARC, LUSC, and LUAD samples were downloaded from The Broad Institute's TCGA data repository. The "genome_wide_snp_6- segmented_scna_minus_germline_cnv_hg19 (MD5)" was used in all datasets. Genome-wide segment counts were summed for each sample according to segment mean data generated based on hg19 reference with germline probes removed. Log2 transformed normalized PLK1 expression and Overall survival (OS) information was downloaded from UCSC Xena platform (https://www.biorxiv.org/content/10.1101/326470v3). Gene expression between normal and tumor were compared with two-tailed unpaired t test under unequal variance assumption. Pearson correlation analysis was used to evaluate association between PLK1 expression and genome segmentation counts. Cutoff finder was used to decide the optimal split for segment count and PLK1 expression to expect survival significance (http://molpath.charite.de/cutoff/). Statistical Package for the Social Sciences (SPSS) software was

used to plot Kaplan-Meier survival curves and to perform log-rank test. GraphPad Prism was used to create box plots and scatter plots."

References

1. Sinha M, Lowell C. Isolation of Highly Pure Primary Mouse Alveolar Epithelial Type II Cells by Flow Cytometric Cell Sorting. Bio-Protocol, LLC; 2016.

2. Wang Y, Zhang Y, Yang J, Ni X, Liu S, Li Z, et al. Genomic Sequencing of Key Genes in Mouse Pancreatic Cancer Cells. Curr Mol Med; 2012