

Figure legends

Supplementary Fig. 1:

a,b, Scatter plots showing linear regression analysis of the correlation between relative *CDCA3* mRNA levels and the number of chromosome arm gains and losses in ADC (**a**) and SqCC cancers (**b**). *R* and *P* values determined according to Spearman's rank correlation. **c,d**, Violin plots showing relative *CDCA3* mRNA levels in diploid and aneuploid ADC (**c**) and SqCC cancers (**d**). **e,f**, Violin plots showing relative *CDCA3* mRNA levels in ADC (**e**) and SqCC cancers (**f**) exhibiting no whole-genome doublings, a single whole-genome duplication or two whole-genome doublings. **c-f**, *P* values as shown determined using unpaired Student's *t* test. **g**, *Left*, dose response curves for eight NSCLC cell lines treated with escalating doses of cisplatin. *Middle*, dose response curves for seven NSCLC cell lines treated with escalating doses of carboplatin. Cells were treated with cisplatin or carboplatin for 48 hours before assessing cell viability. *Right panel*, Cisplatin and carboplatin potency values (IC_{50}) were calculated using GraphPad Prism and listed for each cell line. *n* = 4 (cisplatin), *n* = 3 (carboplatin). **h**, Cells endogenously expressing high versus low *CDCA3* levels were untreated, cisplatin treated for 12 h (cisplatin) or cultured in fresh growth medium for 8 h following cisplatin treatment (recovery). Data points represent an average of γ H2AX foci/nuclei per field of view from a minimum of 800 nuclei (*n* = 13-16 fields). Blue lines indicate median values. Dotted black lines highlight change in foci count following recovery. Percent recovery calculated by difference between 100% and ratio of recovery/cisplatin expressed as a percentage. **i**, Representative CellProfiler software images indicating detection of nuclei (*left panel*), foci (*middle panel*) and merged foci within detected nuclei (*right panel*).

Supplementary Fig. 2:

a,b, Amino acid sequence analysis of the full length *CDCA3* to predict the presence of disordered regions (PrDOS, **a**) and secondary structures (PSIPRED, **b**). **a**, Ordered and disordered predictions are overlaid the full length *CDCA3* sequence with legend describing predictions listed within figure (*lower right*). **b**, Secondary structure predictions indicated above each amino acid of the full length *CDCA3* sequence. Legend describing predicted secondary structures listed in figure (*lower right*). C, coil; H, helix.

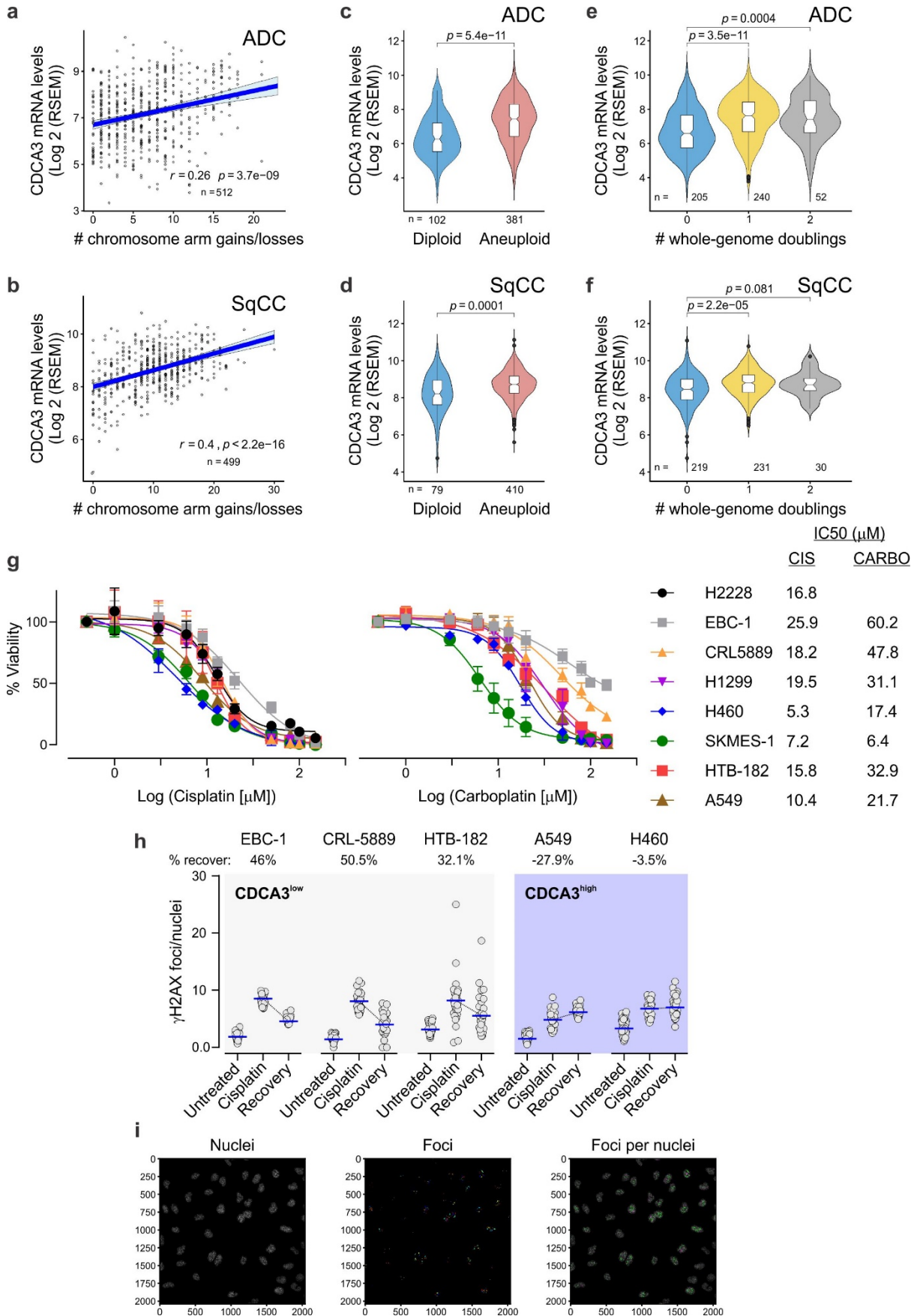
Supplementary Fig. 3:

a, *CDCA3* protein levels determined by immunofluorescence and high throughput microscopy of cells transfected with control siRNA or 62 siRNAs targeting expression of 38 different serine/threonine protein kinases. Microscopy was performed 36 hours following transfection. Protein levels were determined using CellProfiler software analysis and reported as normalised to siRNA control cells. A minimum of 1000 cells were analysed. **b**, Endogenous *CDCA3* western blot analysis of eight NSCLC cell lines treated with or without CX-4945 for 12 hours. Phosphorylated CK2 substrate probe to assess impact of CK2 inhibition with CX-4945. Tubulin was used as a loading control.

Supplementary Fig. 4:

a, Western blot analysis of lysates from CRL-5889, EBC-1 and HTB-182 cells transfected with empty vector or CDCA3-FLAG. Transfected cells as used in FLAG and CDCA3 western blot analysis was performed to detect ectopic and endogenous CDCA3 respectively. Tubulin was used as a loading control. *Arrowhead*, ectopic CDCA3. *Asterix*, endogenous CDCA3. Representative western blot analysis from three independent experiments. Corresponds with Fig. 6a,b. **b**, Dose response curves for eight NSCLC cell lines treated with escalating doses of CX-4945. Cells were treated with CX-4945 for 48 hours before assessing cell viability. *Right panel*, CX-4945 potency values (IC_{50}) were calculated using GraphPad Prism and listed for each cell line. n = 4.

Supplemental Figure 1



Supplemental Figure 4

