Göbl, Morris & Van Dam et al. Fig S1

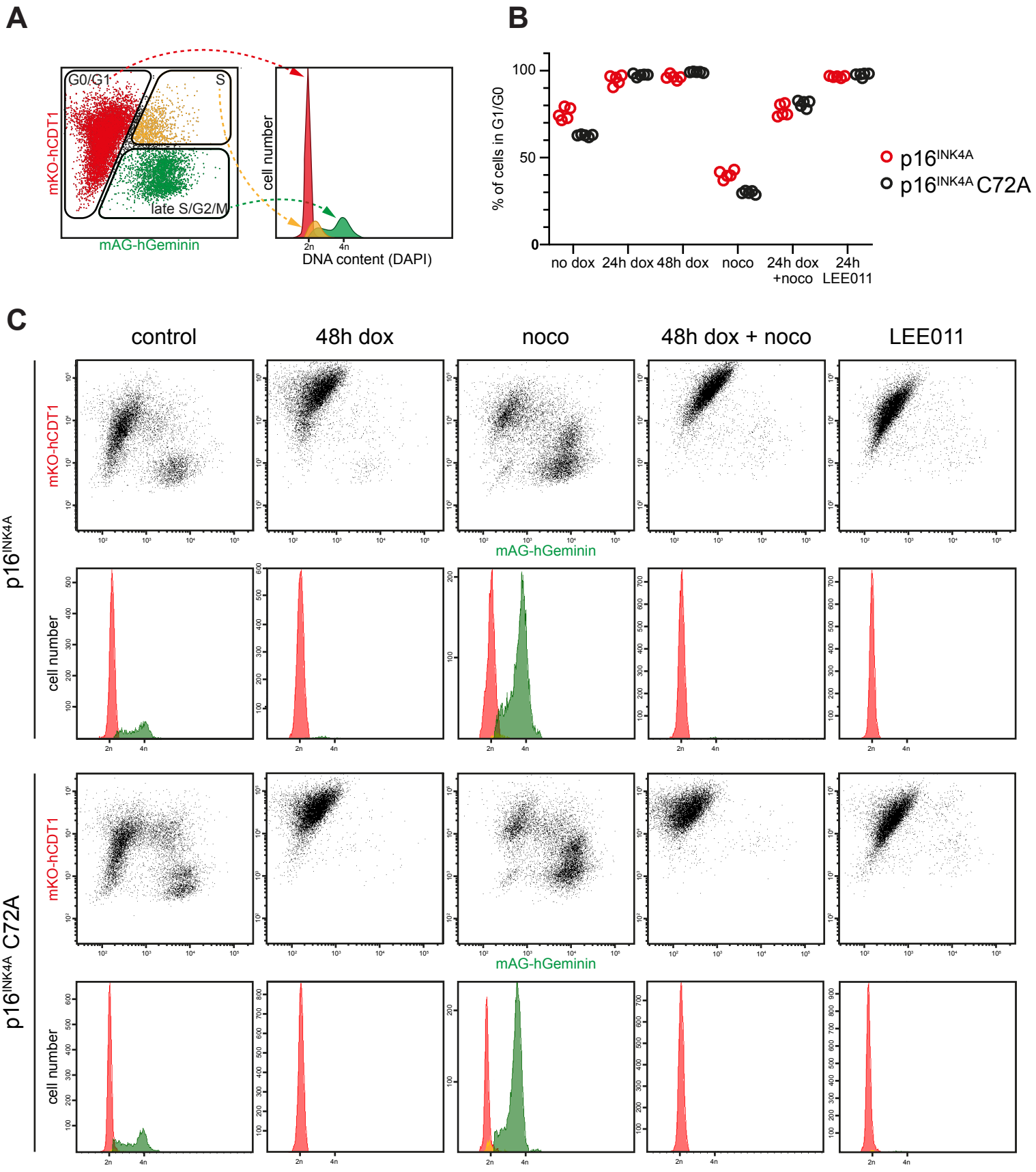

Figure S1. p16^{INK4A} Cys72 is dispensable for its role in cell cycle arrest
(A) Cell-cycle profiling of COLO-829 cells expressing doxycycline inducible pINDUCER20-p16INK4A wildtype or p16^{INK4A}C72A as well as the FUCCI cell cycle indicator system. Cells were trapped overnight in mitosis using Nocodazole to make G1/G0-arrested cells more obvious from those that were still cycling. The CDK4/6 inhibitor LEE011 was added to estimate to what extent CDK4/6 inhibition can induce cell cycle arrest in this cell line. The C72A mutation leaves the cell-cycle inhibitor function of p16^{INK4A} intact. **(B)** The graph shows the pooled results for the percentage of cells in G1/G0 phase of the cell cycle quantified from two experiments with at two or three replicates each. We conclude that p16^{INK4A}C72A is still able to induce a cell cycle arrest. **(C)** The FACS plots and histograms show typical results for each of the conditions.