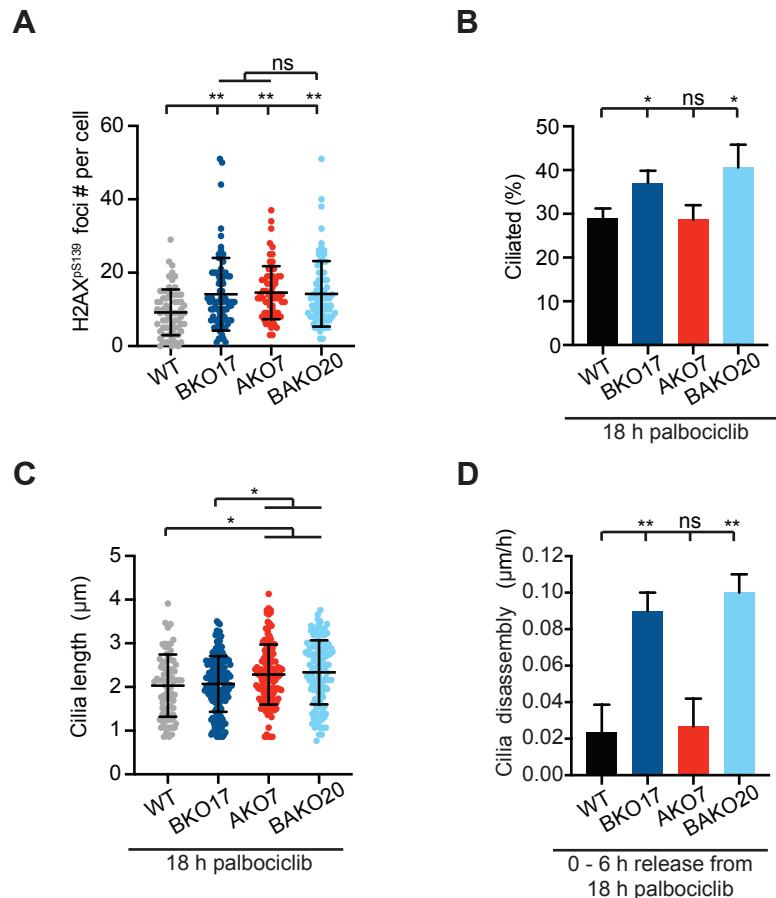


**Figure S1.** *hCDC14A/B* deletion provokes accumulation of DNA damage in cycling cells. **(A)** Cycling cells were stained for the DNA damage marker H2AX<sup>pS139</sup> and analyzed by IF microscopy. Quantification of H2AX<sup>pS139</sup> foci revealed slightly more DNA damage in cells with disrupted *hCDC14A/B* function. N=3, n>90 cells. Mean ± SD. **(B)** Representative cells of quantification from (A).

Data information (A) One-way ANOVA (B) Scale bar: 5 μm



**Figure S2. Confirmation of phenotypes using a different set of *hCDC14A* KO (AKO7), *hCDC14B* KO (BKO17) and *hCDC14A/B* KO (BAKO20) cells. (A)** H2AX<sup>pS139</sup> foci quantification. N=2, n>60 cells. Mean ± SD. **(B, C)** Cilia forma-tion efficiency (B) and cilia length (C) of cells 18 h after palbociclib treatment. N=3, n>150 cells. Mean ± SD. **(D)** Assessment of cilia disassembly rate upon release from palbociclib induced G1 block by calculating cilia length reduction 6h after release of cells from (C) N=3, n>150 cells. Mean ± SD.

Data information (A, C) One-way ANOVA (B, D) T-test