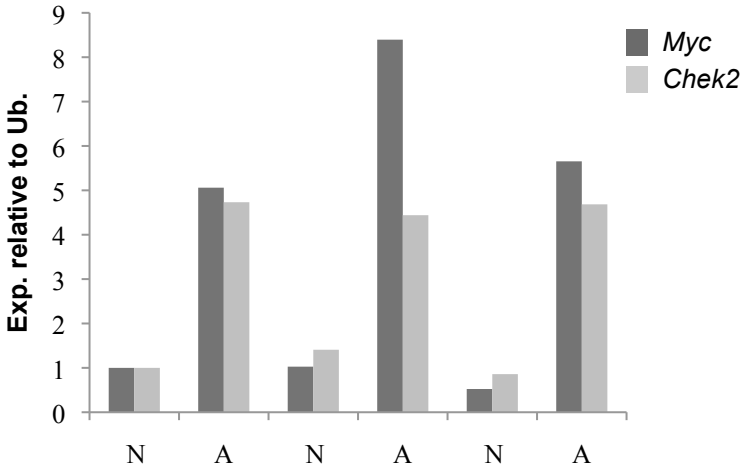
Supplemental Figure 1. Normal tissue or small intestine adenomas from *Apc*^{Min} mice were harvested at around 120 days of age. Tumor material was snap frozen and analyzed by qRT-PCR analysis for expression of *Chek2* and c-*Myc* transcript levels.

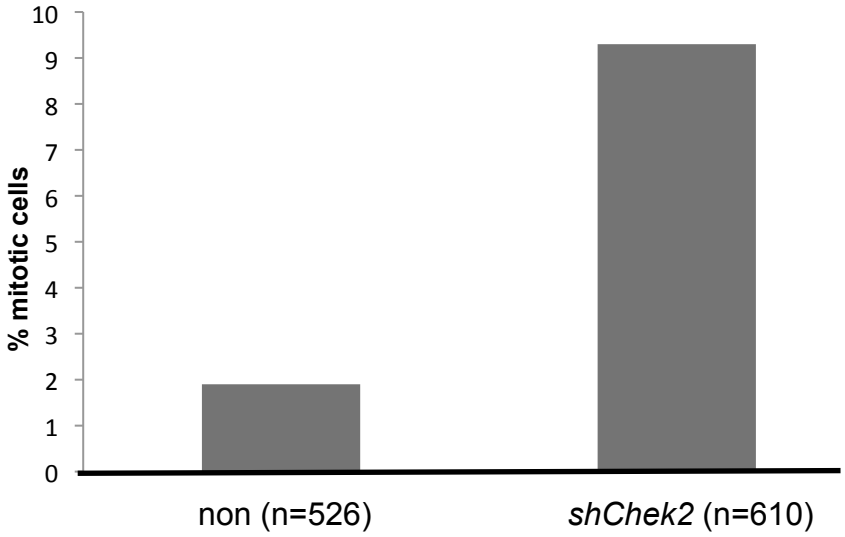
Supplemental Figure 2. NIH-3T3 fibroblasts infected with MSCV-Myc-IRES-GFP retroviruses and shRNA against *Chek2* or a non-target shRNA were stained with antibodies against tubulin and was scored for the amount of cells situated in mitosis. *Chk2* deficient cells have a higher percentage of cells in mitosis (9% vs. 2% for control infected cells).

Supplemental Figure 3. (A) Mouse lymphoma cell line from the λ-*Myc* transgenic mouse was infected with shRNA against *Chek2* or control vector. These cells were then stained with antibodies against tubulin and counter stained with PI. Representative picture of the polyploid (arrow) cells observed in the Chk2 deficient cells. (B) Growth curve of the cells described in (A). Chk2 deficiency severely impacts the division rate of the lymphoma cells. (C) The Chk2 deficient lymphoma cells were also transplanted in recipient CL57/B6 animals and monitored for palpable lymphoma. Disease progression was significantly delayed in mice transplanted with cell lacking Chk2 (Median survival 29 days vs. 17 days of control cells. P = 0.0295). When sick, tumors were snap frozen and analyzed with western blot for expression of Chk2. Tick on survival curve represents a false positive sample harvested.

Supplemental Figure 1

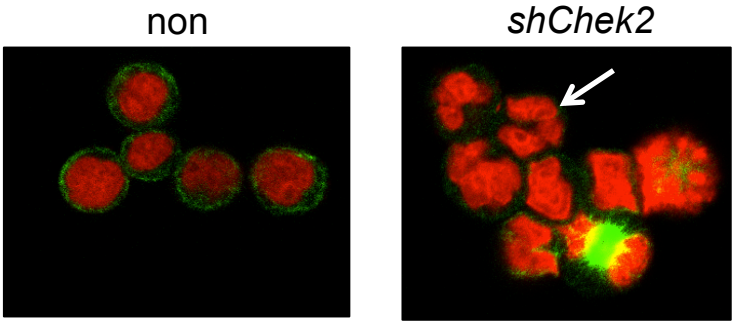


Supplemental Figure 2



Supplemental figure 3

A



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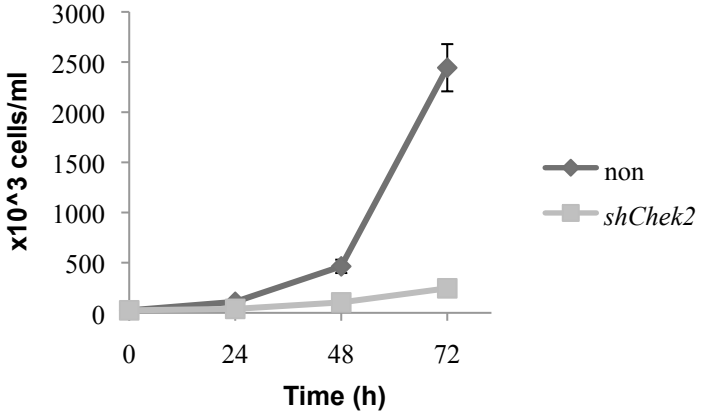

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