## **Supporting Information**

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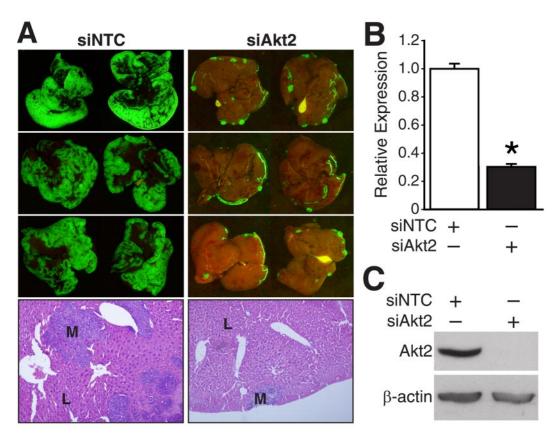


Fig. S1. (A) KM20 cells were transfected with single NTC or Akt2 siRNA duplex *in vitro* and inoculated intrasplenically 56 hours later. Animals were monitored individually for metastatic tumor growth and killed 30 days after intrasplenic injection. Representative photographs of the diaphragmal and visceral liver surfaces are shown. L, normal liver; M, metastasis. (B, C) KM20 cells were transfected with single-sequence NTC or Akt2 siRNA. After 56 hours of incubation, specific knockdown was confirmed by qRT-PCR or Western blot analysis, respectively. β-actin serves as a loading control.

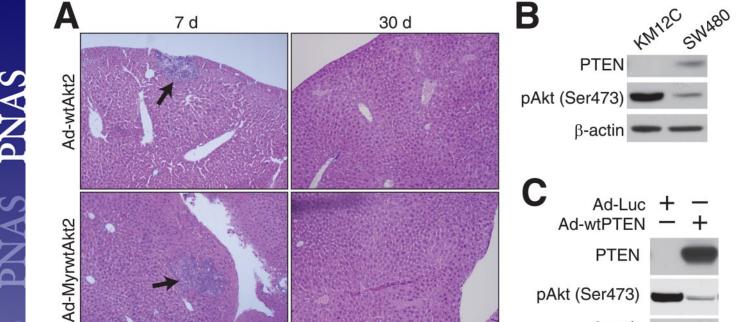


Fig. S2. Initiation of proliferation and loss of metastases. (*A*) SW480 cells were infected with control or wild-type Akt2 adenovirus and injected intrasplenically (n = 3 mice per group). Histological examination of paraffin-embedded liver sections from mice killed on day 7 or day 30 after injection was performed. (*B*) Detection of PTEN and, as a measure of PI3K activation, phosphorylated Akt in KM12C and SW480 cells. *β*-actin serves as a loading control. (*C*) Detection of ectopic PTEN by immunoblot, as well as appropriate inhibition of the PI3K pathway in KM12C cells, as assessed by decrease in phosphorylated Akt. *β*-actin serves as a loading control.

β-actin



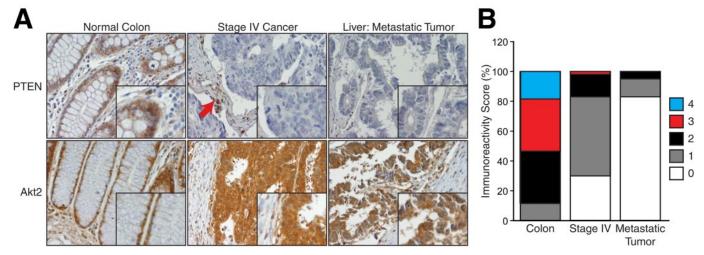


Fig. S3. PTEN expression and localization in colorectal cancer samples. (*A*) Expression of PTEN in representative normal colon, colorectal adenocarcinoma and metastatic liver tumor on tissue microarray (×200 magnification, insert ×400; *n* = 18 cases; 72 tumor cores, 18 non-neoplastic cores). Arrow demonstrates PTEN expression in connective tissue. Akt2 expression in matched tissue samples is shown below. (*B*) Distribution of PTEN immunoreactivity scores in normal mucosa, primary, and metastatic tumors.