Supplemental material and methods:

Cell culture

Cell lines were derived from murine KPC tumors as previously described [1] and maintained in DMEM (41966029, Invitrogen) + 10% FBS (SH30070.03, HyClone). Cancer associated fibroblast were obtained by serial trypsinization of small tumor pieces originating from *LSL-Kras$^{G12D/+}$;Ptf1a-Cre* (KC) mice. Protein lysates were obtained using RIPA buffer with protease and phosphatase inhibitors.[2]

Automated quantification of immunohistochemistry:

Automated quantification of IHC for cleaved caspase-3, phospho-histone H3 and ki67 was performed on 30 randomly chosen fields using the Ariol SL-50 imaging system and analysis software (Leica) as described.[3]

SPARC ELISA:

Human SPARC ELISA Kit was purchased from Aviscera Bioscience Inc. (SK00766-01). Mouse EDTA plasma samples were diluted and compared to reference standards and processed according to the manufactures instructions. The lower limit of quantification for this assay was 0.128ng/ml.

Full blood counts:

Full blood counts were obtained on terminal cardiac bleeds using a MS9-5 Veterinary Hematology Analyzer (Woodley Equipment Company Ltd.)

Supplementary references:

1. **Hingorani SR**, Wang L, Multani AS, et al. Trp53R172H and KrasG12D cooperate to promote chromosomal instability and widely metastatic pancreatic ductal adenocarcinoma in mice. *Cancer Cell* 2005;**7**:469-83.
2. **Karreth FA**, DeNicola GM, Winter SP, et al. C-Raf inhibits MAPK activation and transformation by B-Raf(V600E). *Mol Cell* 2009;**36**:477-86.
3. **Frese KK**, Neesse A, Cook N, et al. nab-Paclitaxel potentiates gemcitabine activity by reducing cytidine deaminase levels in a mouse model of pancreatic cancer. *Cancer Discov* 2012;**2**:260-9.