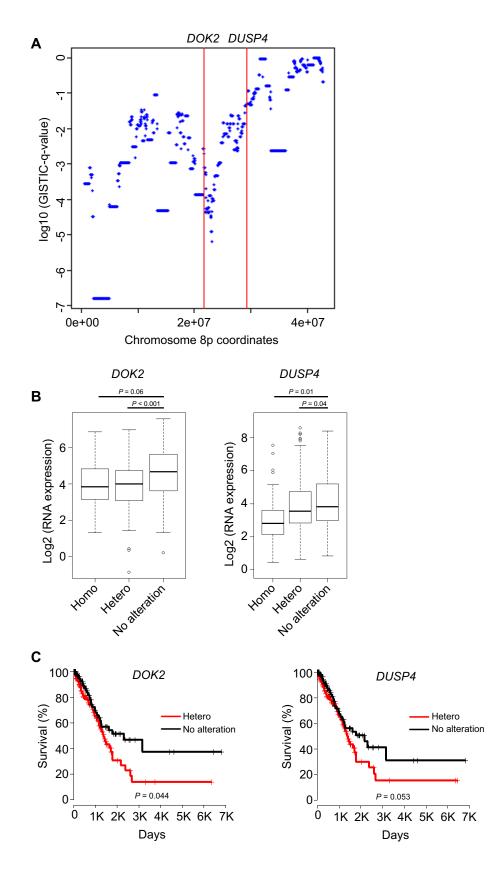


Supplemental Figure 1. Histopathology of lung tumors in $Dok2^{+/-}Dusp4^{+/-}$ mice. (A) H&Estained adenocarcinoma from lung tissue of two $Dok2^{+/-}Dusp4^{+/-}$ mice at 9 months of age. Scale bar, 50 µm. (B) H&E-stained adenocarcinoma with papillary features (*) and solid growth areas (**) from a $Dok2^{+/-}Dusp4^{+/-}$ lung. Middle, close-up of solid growth region. Right, close-up of papillary growth region. Scale bar, 50 µm. (C) Serial sections of lung tissue stained with H&E and IHC for phosphorylated Erk and Ki67 from 18-month od WT and $Dusp4^{+/-}$ mice. Scale bars, 50 µm. Insets show the staining from enlarged normal lung tissues.

Chen et al., Suppl. Fig 2



Supplemental Figure 2. Compound loss of *DOK2* and *DUSP4* expression and its clinical implication in human lung adenocarcinomas. (A) Size and extent of chromosome 8p deletions (in blue) from individual lung adenocarcinomas based on aCGH data analysis. Red line indicates genomic position of *DOK2* and *DUSP4*. (B) A plot shows the correlation of copy number and gene expression level for *DOK2* and *DUSP4* in lung cancer samples. (C) The comparison of overall Kaplan-Meier survival curve between lung adenocarcinoma patients with no alteration versus heterozygous loss for *DOK2* or *DUSP4*. *P* values were determined by log-rank test.

