

Supporting Information

A Panel of Protein Kinase Chemosensors Distinguishes Different Types of Fatty Liver Disease

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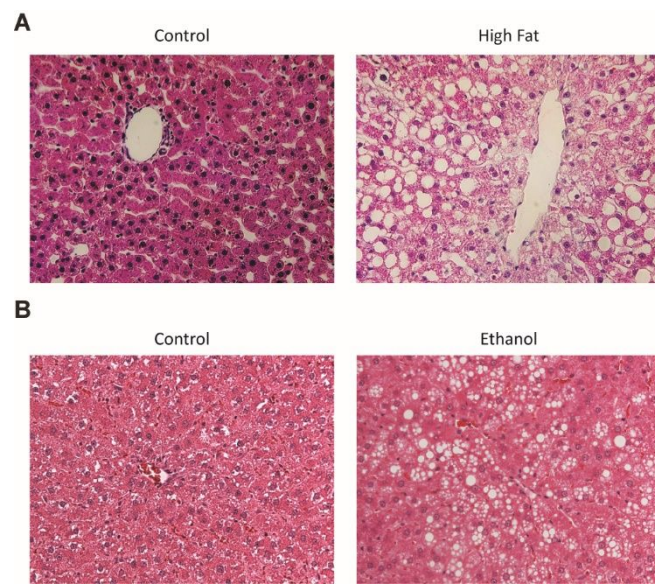
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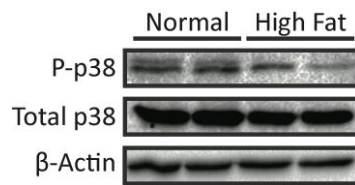
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Figure S1



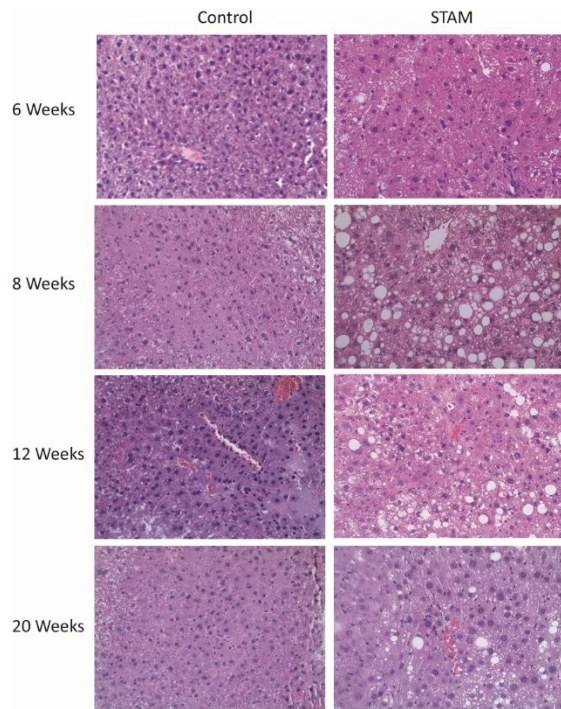
Indistinguishable histology of NAFLD and AFLD. H&E staining for NFALD (A) and AFLD (B) livers.

Figure S2



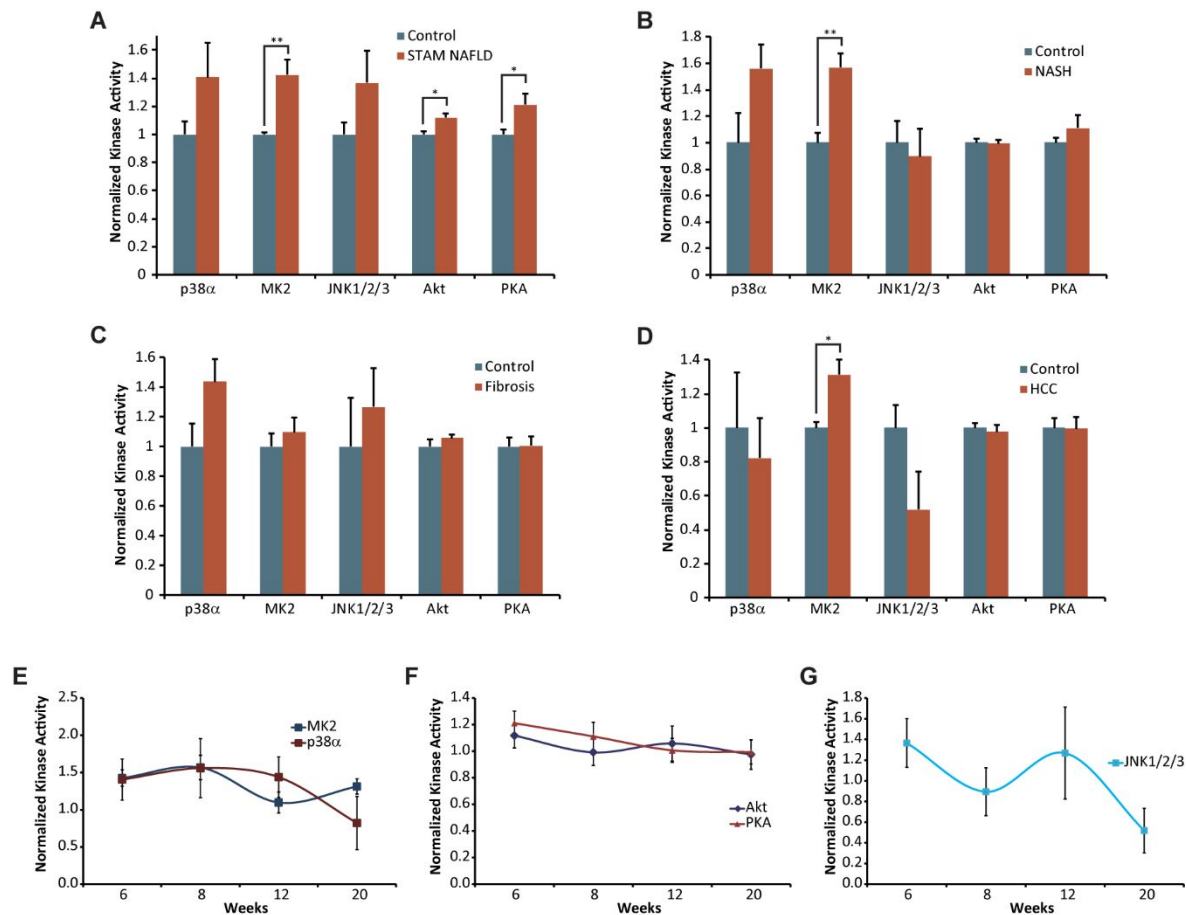
NAFLD livers display decreased phosphorylation of p38. Reduced phosphorylation of the p38 (P-p38) activation loop is observed in NAFLD (High Fat) versus control (Normal) livers. Total p38 staining indicates that levels of p38 do not change appreciably in each sample. Total p38 and P-p38 antibodies recognize α , β , γ , and δ isoforms of p38. β -actin staining demonstrates equivalent protein loading in each lane.

Figure S3



Progression of the STAM model through increasingly severe stages of liver disease. H&E staining of livers from animals fed a control diet as well as STAM animals at varying time points corresponding to NAFLD (6 weeks), NASH (8 weeks), fibrosis (12 weeks), and HCC (20 weeks).

Figure S4



Changes in kinase activity in the STAM model. Kinase activities for STAM NAFLD (A, $n = 5$), NASH (B, $n = 5$), fibrosis (C, $n = 5$), and HCC (D, $n = 5$) relative to control animals ($n = 5$) are shown. Significant increases in MK2 (43%), Akt (12%), and PKA (21%) activities are observed in STAM NAFLD. A significant increase in MK2 activity in NASH (57%) and HCC (31%) was observed. Kinase activities for MK2 and p38α (E), Akt and PKA (F), and JNK (G) are clustered according to similar trends in activity during disease progression. STAM ($n = 5$) samples are normalized to control animals ($n = 5$). Activities are shown versus time for STAM NAFLD (6 week), NASH (8 week), fibrosis (12 week), and HCC (20 week) samples. P-values from a two-tailed t-test are represented by * (< 0.05) or ** (< 0.01). Error bars represent sem.