Supporting Information Figure 1: Majority of EYFP +ve cells in the liver are F4/80+ve and CD11b+ve.

(A) FCM analysis of cells isolated from the liver is showing that the majority of EYFP+ cells in the liver are CD11b+. (B) Quantification of the number of F4/80+/ EYFP+ cells in the liver, showing the majority of EYFP+ cells in the liver (C) are F4/80+ scale bar 50 μ m. (D) Double-staining for EYFP and BrdU is showing no EYFP+ cell which is positive for BrdU in Sham and burn group (day 7) and a very limited number of EYFP+ which is positive for BrdU at day 14 post-injury. The arrow shows proliferating EYFP +ve cell; arrowhead shows proliferating EYFP –ve cell; scale bar 100 μ m. Note that the majority of EYFP+ cells are BrdU negative. Please note that the small red particles which are not pointed out are autofluorescence most probably due to the presence of red blood cells.

Supporting Information Figure 2: Thermal injury increases the percentage of bone marrow myeloid cells.

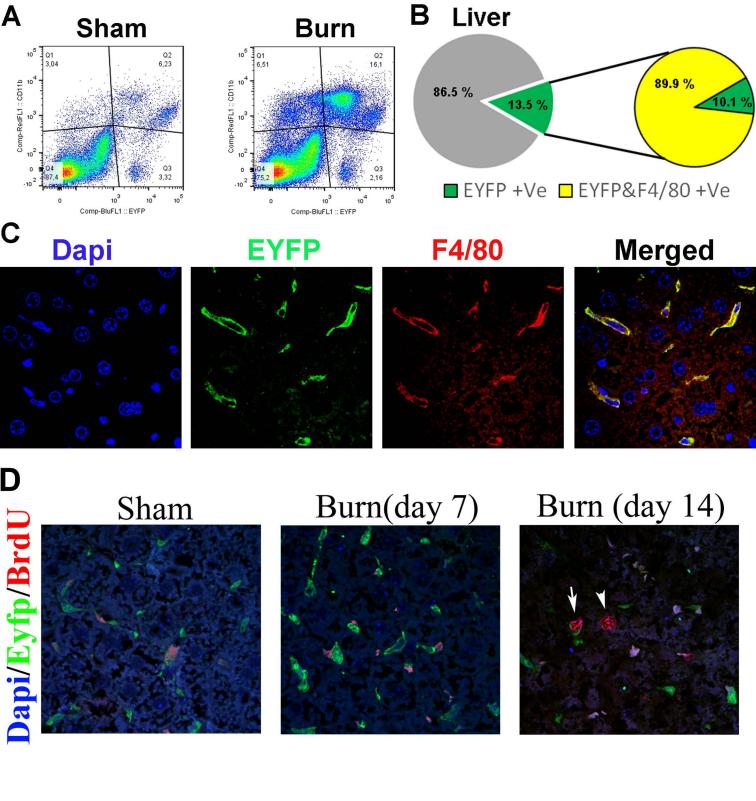
(**A and B**) Flow cytometry on bone marrow-derived cells shows a 50% increase in the percentage of EYFP +ve cells post-thermal injury.

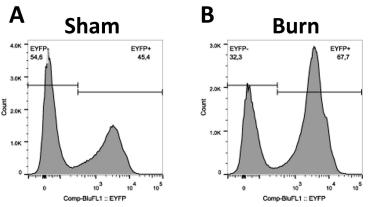
Supporting Information Figure 3: Thermal injury up-regulates several pathways that associated with liver injury in the myeloid cells.

(A) There is an up-regulation of genes in EYFP +ve cells associated with liver dysfunction (B) Pathway analysis shows high up-regulation of the coagulation system in the myeloid cells.

Supporting Information Figure 4: EYFP+ve cells are skewed more towards a proinflammatory phenotype 1-week post thermal injury *in vivo*. (A) Heat map of upregulated genes associated with inflammation and wound healing in EYFP+ cells from the livers of sham and thermally injured mice. (B) Measurement of liver function enzymes ALT and AST show Ketanserin is not having a detrimental effect on the liver's functional capacity. (C) Ketanserin does not modulate all the genes, which are associated with pro-inflammatory macrophages. (D) qPCR analysis of relative mRNA expression of M1 and M2 associated genes of BMDM from thermally injured mice treated with vehicle or Ketanserin. (*P<0.05).

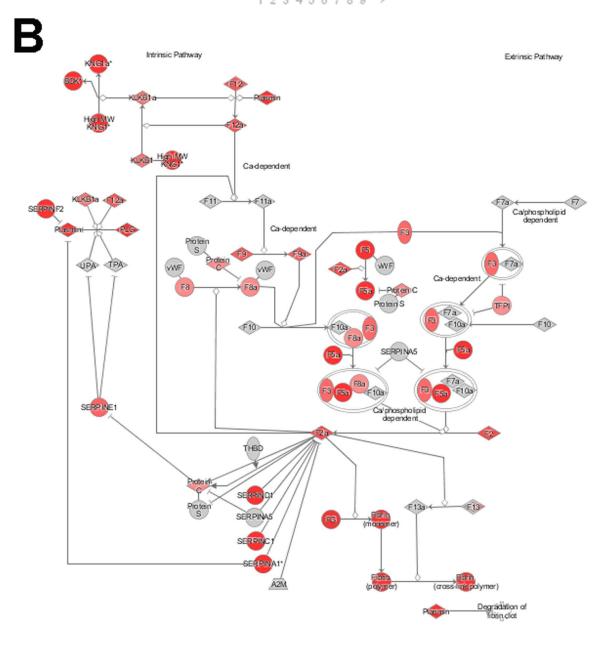
Supporting Information Figure 5: Pathway analysis shows deregulation of numerous pathways associated with inflammation and fibrosis.

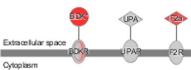


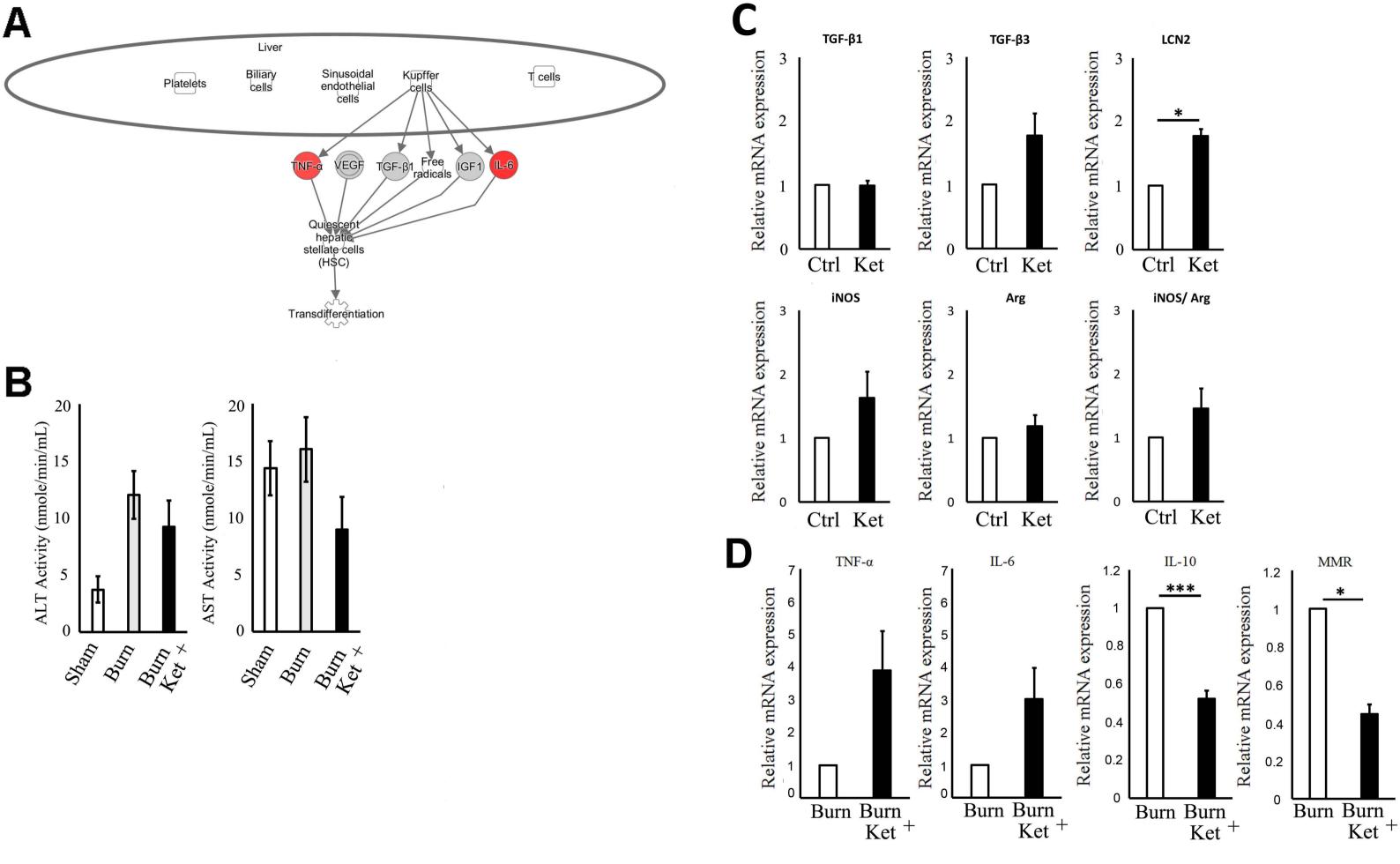


▲ V Hepatotoxicity

Name	p-value range		# Molecules
Liver Necrosis/Cell Death	***	3.07E-01 - 1.82E-09	55
Liver Steatosis	** • • • •	3.07E-01 - 4.07E-09	58
Liver Inflammation/Hepatitis	SUN	3.84E-01 - 9.39E-09	57
Liver Damage	poper i	4.74E-01 - 3.17E-08	56
Liver Cholestasis	1111111111	3.07E-01 - 6.41E-08	28







Granzyme A Signaling

Dendritic Cell Maturation

Noradrenaline and Adrenaline Degradation

Mineralocorticoid Biosynthesis

Inhibition of Matrix Metalloproteases

Trans, trans-famesyl Diphosphate Biosynthesis

Phenylalanine Degradation I (Aerobic)

Autoimmune Thyroid Disease Signaling

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Type I Diabetes Mellitus Signaling

VDR/RXR Activation

B Cell Development

a-tocopherol Degradation

Thyroid Hormone Metabolism II (via Conjugation and/or Degradation)

Superpathway of Geranylgeranyldiphosphate Biosynthesis I (via Mevalonate)

0.0

2.5

5.0

7.5

10.0

12.5

-log(p-value)

15.0

15

37

30

97

20.0

17.5

22.5