Supporting Materials and Methods

Adeno-associated virus vector

We used a two-plasmid calcium phosphate transfection system to produce replication-defective rAAV, rAAV8.pAlb.mCherry.hDTR. The transgene-encoding plasmid, pAAV.pAlb.mCherry.T2A.hDTR, was constructed by inserting DNA encoding mCherry into the SalI and BamHI sites, a T2A peptide linker sequence into the BamHI and SacII sites, and DNA encoding the human diphtheria toxin receptor (hDTR; provided by Richard Palmiter, University of Washington) into the SacII and XhoI sites of the base pAAV.pAlb plasmid. The base pAAV.pAlb plasmid was constructed by inserting AAV serotype-2 inverted terminal repeats into the BglII and NdeI restriction sites in pLIVE plasmid (Mirus Bio, Madison, WI), which has the multiple cloning site under the control of the minimal mouse albumin promoter. The pDG2/8 helper plasmid contained AVV serotype-2 replication components and AAV serotype-8 capsid protein (pDG base plasmid provided by Dr. Jurgen Kleinschmidt, Deutsches Krebsforschungszentrum Stiftung des offentlichen Rechts and serotype-8 capsid DNA provided by Dr. James Wilson, University of Pennsylvania).

For production of rAAV, human epithelial kidney 293s were grown in DMEM (high glucose, +L-glutamine; Gibco/Thermo Fisher Scientific) + 10% Cosmic Calf Serum (Hyclone Laboratories, Inc, Logan, Utah) + 1% Penicillin/Streptomycin (Gibco/Thermo Fisher Scientific), were transfected with 10 µg pDG82/8 plasmid to 2.36 µg pAAV.pAlb.mCherry.T2A.hDTR, and grown at 37°C, 5-7.5% CO_2. Media was replaced the following day with DMEM (high glucose, +L-glutamine) + 1% Penicillin/Streptomycin/Glutamine (Gibco/Thermo Fisher Scientific). After 72 hours, the cells were lysed by 3 freeze-thaw cycles, incubated with 10 U/µL DNAse, pelleted, the supernatant purified by an iodixonal gradient (Optiprep; Sigma-Aldrich), and desalted

using an Amicon Ultra-15 100K centrifugal filter device (EMD Millipore, Bedford, MA) with 1X PBS. The AAV was stored in 5% glycerol at -80°C. Quantification was done using the QuickTiter AAV Quantitation Kit, according to the manufacturer's instructions (Cell Biolabs, San Diego, CA).

In vivo cell depletion

For both monocyte and neutrophil depletion, depletion antibodies were injected intraperitoneally 24 hours before, at the time of, and 24 hours after DT injection in 200 µL 1X PBS. Anti-CCR2, Clone MC-21 (gift from Dr. Matthias Mack, Universitätsklinikum Regensburg) and *InVivo*Plus Rat IgG2b Isotype Control, Clone LTF-2 (BP0090, Bio X Cell, West Lebanon, NH) were dosed at 45 and 50 µg/mouse/day, respectively (1). *InVivo*Plus anti-mouse Ly6G, Clone IA8 (BP0075-1, Bio X Cell) and *InVivo*Plus Rat IgG2a Isotype Control, Clone 2A3 (BP0089, Bio X Cell) were dosed at 250 µg/mouse/day.

APAP-Induced Liver Injury

Mice were fasted overnight prior to IP injection of 300 mg/kg-bw APAP (gift from Dr. Terrence Kavanagh, University of Washington) in 0.9% saline, exact volume varied depending on mouse weight (10 µL/g-bw); however, injection volume was approximately 400 µL. Mice also received an IP injection of 200 µL 1X PBS at the time of APAP injection. Mice were anesthetized for liver perfusion at 6 and 24 hours post treatment.

Liver perfusions and isolation of liver cells

Briefly, mice were anesthetized with Avertin, the portal vein cut, up to 500 µL of blood collected for serum analysis of ALT and protein, and ~2/3 of the right posterior lobe cut off, with a piece for RNA put into TRIzol and a piece for histology put into 10% neutral-

buffered formalin and fixed for approximately 24 hours. Perfusion solution followed by approximately 8 mL of 0.8 mg/mL collagenase solution was pumped through the liver, the liver cut out, and gently mashed in Wash Buffer (1X PBS + 4% FBS (heat-inactivated; Corning, Corning, NY and Gibco/Thermo Fisher Scientific) to form a single cell suspension. The hepatocyte pellet was washed two times with Wash Buffer and 50 µL taken into TRIzol for RNA analysis. The non-parenchymal cells contained in the supernatant were isolated on a 20% iodixanol layer and resuspended in Flow Buffer (1X PBS + 2% FBS + 1 mM EDTA) for staining.

Flow cytometry and cell sorting

Isolated non-parenchymal cells were stained with the following antibodies from BioLegend (San Diego, CA): anti-IA/IE (1:1000, M5/114.15.2, Cat. No. 107606), anti-Tie2 (1:400, TEK4, Cat. No. 124008), anti-Ly6C (1:800, HK1.4, Cat. No. 128012), anti-F4/80 (1:800, BM8, Cat No. 123114), and anti-Ly6G (1:400, 1A8, Cat No. 127624); and from BD Biosciences (San Jose, CA): anti-CD11b (1:400, M1/70, Cat No. 563015) and anti-CD11c (1:400, HL3, Cat. No. 562454). Cells were also stained with LIVE/DEAD Fixable Far Red Dead Cell Stain Kit (1:1000, Molecular Probes/Thermo Fisher Scientific, Waltham, MA). Cells were isolated by fluorescence-activated cell sorting using a BD Aria III (BD Biosciences). Analysis of cell populations was performed using FlowJo software, version 9.8.5 (FlowJo, LLC, Ashland, OR). The purity of isolated cell populations was measured based on the expression of cell-specific genes (Supporting Figure 7).

Quantitative RT-PCR gene expression analysis

Liver tissue, hepatocytes, LSECs, KCs, and Huh7, and Huh7-TLR3 cells were stored in TRIzol at -80°C until processing. Liver samples were homogenized and hepatocyte samples were sheared by passage through a 27G1/2 needle 3-5 times. All samples

were treated with proteinase K (Qiagen, Valencia, CA) for 20 min at 56°C. RNA was isolated using the Direct-zol RNA Miniprep Kit (Zymo Research Corporation, Irvine, CA). Complementary DNA was generated using the QuantiTect Reverse Transcription Kit (Qiagen), preamplified using BIO-X-ACT Short Mix (Bioline USA Inc., Taunton, MA) and the TaqMan assays of interest (Applied Biosciences/Thermo Fisher Scientific) (Supporting Table 1), and diluted 1:5 for analysis. Microfluidic quantitative RT-PCR was performed on a BioMark HD microfluidics system (Fluidigm, South San Francisco, CA). The Fluidigm Gene Expression software was used to calculate Ct thresholds and Excel used to calculate relative expression levels and fold change values using the $2^{-\Delta Ct}$ and $2^{-\Delta\Delta Ct}$ methods, respectively. Relative expression was calculated relative to *Hprt* in mice and *HPRT in Huh7 cells*. For mouse studies, fold change was calculated as the ratio between individual mice compared to the median of the mice in the + PBS group. Except in the time course experiment where raw Ct values from two experiments were combined and analyzed together, fold change values were calculated per experiment and graphed in sum.

Measurement of protein

Blood collected from the portal vein was allowed to clot at room temperature for a minimum of 30 min and then centrifuged at 6000 rpm, for 10 min, and the supernatant collected. When necessary, the supernatant was clarified for an additional 4 min at 6000 rpm. Levels of serum ALT were quantified using the Alanine Aminotransferase-SL Kit (Sekisui Diagnostics LLC, Lexington, MA). Levels of CCL-2 (MCP-1), IL-1β, IL-6, CXCL1, IL-10, and TNF-α were quantified using the Mouse MCP-1 Ultra-Sensitive Kit and a Custom Mouse Cytokine V-PLEX Kit, according to the manufacturer's instructions (Meso Scale Diagnostics, LLC, Rockville, MD). Analysis was performed in the Discovery Workbench 3.0 software (Meso Scale Diagnostics, LLC).

Western blotting

Mouse hepatocyte, Huh7, or Huh7-TLR3 pellet was resuspended in RIPA Buffer (with Thermo Halt Protease Inhibitor Cocktail and EDTA; Thermo Fisher Scientific) and protein was quantified with the BioRad DC Protein Assay (Bio-Rad Laboratories, Hercules, CA), according to the manufacturer's instructions. For mouse hepatocyte samples, 20 µg protein/well was resolved on 12% SDS-PAGE gels; and for Huh7 and Huh7-TLR3 samples, 50 µg protein/well was resolved on 10% SDS-PAGE gels. All gels were transferred onto Immobilon-P PVDF membrane (EMD Millipore Corporation, Darmstadt, Germany) and dried before further processing. For detection of hDTR and hTLR3, blots were rewet in 100% methanol, blocked for 1 hour at room temperature, incubated with either anti-hHB-EGF (1:2,000; AF-259-SP, R&D Systems, Minneapolis, MN) or anti-TLR3 (1:1,000; NBP2-24875, Novus Biologicals, received as a gift from Dr. Stephen Polyak) in blocking solution overnight at 4°C, washed in 0.1% PBST, incubated with either Donkey anti-Goat IgG (H+L) (1:20,000; A16005, Invitrogen/Thermo Fisher Scientific) in blocking or goat anti-mouse HRP (1:20,000; Bio-Rad Laboratories) in 0.1% PBST for 2 hours at room temperature, and washed with 0.1% PBST followed by 1X PBS. Blocking solution for hDTR was 5% donkey serum (D9663, Sigma-Aldrich) in 0.1% PBST and for hTLR3 was 5% nonfat milk in 0.1% PBST. For detection of mCherry and β-actin, blots were processed dry, incubated with either anti-mCherry (1:1000; ab167453, Abcam, Cambridge, UK) or anti-β-actin (1:1000; ab81227, Abcam) in 5% nonfat milk in 0.05% PBST for 1 hour at room temperature, washed with 1X PBS, incubated with goat anti-rabbit HRP (1:20,000; 1705046, Bio-Rad Laboratories) for 30 min at room temperature, and washed with 1X PBS. Blots developed in Western Lightning Plus-ECL (PerkinElmer, Waltham, MA) according to manufacturer's

instructions and exposed to Amersham Hyperfilm ECLfilm (GE Healthcare, Little Chalfont, UK).

Histology

Liver pieces were fixed in 10% neutral-buffered formalin for approximately 24 hours and then stored in 1X PBS. Samples were either paraffin-embedded, sectioned, and stained with hematoxylin and eosin; or OCT-embedded, cryo-sectioned, and stained with DAPI. All images were taken on a Nikon Eclipse T*i*-E inverted microscope with a Nikon Digital Sight DS-Ri1 camera using NIS-Elements, version 4.50, Advanced Research software (Nikon, Melville, NY), at room temperature. Both the 10X objective (0.30 NA) for a total 100X magnification and the 20X objective (0.45 NA) for a total of 200X magnification were used. Fluorescent image acquisition was conducted with consistent exposures per channel to facilitate cross-image conformity. Fluorescent images are the merge of 3 channels: FITC for hepatocyte auto-fluorescence false-colored green, PETR for mCherry fluorescence false-colored red, and UV for DAPI nuclear staining false-colored blue. Image processing was performed in NIS-Elements, version 4.3 software (Nikon). All embedding, sectioning, and staining was performed by the University of Washington Histology Imaging Core.

Cell Lines

Huh7 and Huh7-TLR3 human hepatoma cell lines were kindly provided by Dr. Stephen Polyak (University of Washington). Cell lines were maintained in DMEM (high glucose, +L-glutamine) + 10% FBS + 1% Penicillin/Streptomycin, all from Gibco/Thermo Fisher Scientific. Huh7-TLR3 media also contained 2 µg/mL Blasticidin (Gibco/Thermo Fisher Scientific). For determination of mechanism of DT-induced death, Huh7-TLR3 cells were seeded at a density of 20,000 cells/well in a 96 well plate and cultured overnight. Cells

were treated for 12 and 24 hours with 3 ug/mL DT in 1X PBS, 400 nM staurosporine (Fisher Scientific/Thermo Fisher Scientific) in DMSO, 1000 uM hydrogen peroxide (Sigma-Aldrich) in 1X PBS, 1 uM nigericin (ApexBio, Houston, TX) in DMSO, and 1X PBS and DMSO vehicle controls matched to the concentration of DT and staurosporine, respectively. Viability and caspase activation were measured using the ApoTox-Glo Triplex Assay according to the manufacturer's instructions (Promega, Madison, WI). The cytotoxicity measurement was excluded for our purposes as the fluorescent excitation/emission spectrum of Huh7-TLR3 hepatoma cells significantly overlaps with that of the cytotoxicity assay. For measurement of gene expression in DT-killed cells, Huh7 cells were seeded at a density of 50,000 cells/well in a 24 well plate and cultured overnight. Cells were treated for 48 hours with 0, 1, and 100 ng/mL DT in 1XPBS and then two wells of each treatment pooled for RNA analysis.

Statistical analysis

Graphs present data from single experiments or aggregate data from 2-3 independent experiments, exact numbers and repetitions are noted in the figure legends. For all experiments except the C57BL/6J time course, mice in the + DT groups that expressed *hDTR* levels comparable to those in + PBS groups at the 48 hour collection point—indicating failure of DT to reach the liver—were eliminated from the analysis.

Due to the variety range of responses seen in mice, data cannot be assumed to be normal, therefore, nonparametric statistical analyses were performed. For time course data, significance was determined by a Kruskal-Wallis test, followed by pairwise comparisons to the control group using Dunn's post-test. For data comparing wild type (WT) and knock-out (KO), control (+PBS) and treated (+DT) groups, initial p-values were calculated using a pairwise Mann-Whitney test for four comparisons (WT+PBS versus

WT+DT, KO+PBS versus KO+DT, WT+PBS versus KO+PBS, and WT+DT versus KO+DT) and then adjusted p-values calculated using the Holm-Bonferroni method to correct for multiple comparisons. For data comparing monocyte and neutrophil depletion groups, initial p-values were calculated using a pairwise Mann-Whitney test for three comparisons and then adjusted p-values calculated using the Holm-Bonferroni method to correct for multiple comparisons. Correlation plots were evaluated by Goodness of Fit and the R^2 value and significance level provided on the graph. All other data were analyzed using pairwise Mann-Whitney tests. Significant p-values are represented by asterisks at the following levels: *, $P \leq 0.05$; **, $P \leq 0.01$; ***, $P \leq 0.001$; and ****, $P \leq 0.0001$; while ns = not significant ($P > 0.05$). In general, if a test was non-significant it was not represented in the figures. All statistical analysis was performed in either GraphPad Prism 6 (La Jolla, CA) or Microsoft Excel (Microsoft, Redmond, WA).

Supporting Materials, Figure Legends

Supporting Figure 1. Mechanism of DT-induced hepatocyte death. (A) Huh7-TLR3 cell viability and caspase 3/7 activation following 12 and 24 hours of treatment with DT, staurosporine (apoptosis inducer), hydrogen peroxide (necrosis inducer), nigericin (pyroptosis inducer), or vehicle control. PBS is the control for DT and hydrogen peroxide, indicated by an "*" and DMSO is the control for staurosporine and nigericin, indicated by a "$^{+}$". Data are representative of two plates run in parallel at each time point, each bar represents the mean of triplicate measurements. (B) Serum ALT levels in rAAV-transduced B6 and RIPK3$^{-/-}$ mice 48 hours following DT injection. Each data point represents an individual mouse (n = 4-6 per group), bars represent the median. Significance determined by a pairwise Mann-Whitney. DT, diphtheria toxin; H2O2, hydrogen peroxide; RIPK3, receptor-interacting protein kinase 3; and ns, not significant.

Supporting Figure 2. Differential gene expression in hepatocyte-specific and APAP-induced liver injury mouse models. Fold change gene expression in total liver of (A) rAAV-transduced C57BL/6J mice 48 hours following DT administration (n = 6) compared to the median of the No DT group (n = 4) and (B) C57BL/6J mice 24 hours following APAP and PBS administration (n = 5) compared to the median of historical control C57BL/6J mice that received no treatment (n = 6). *Hprt* used as reference gene. Each column represents an individual mouse. (C) Serum ALT, (D) monocyte, and (E) neutrophil numbers were measured in APAP-treated mice 6 and 24 hours post treatment (n = 5 per group). (A) Data combined for 2 independent experiments, some of the assays are also represented in Figure 3 as bar graphs. The "*" indicates that the median of the No DT group only consisted of 1 mouse as *Ifnb1* was not detected in 3 of 4 mice.

Supporting Figure 3. Preliminary gating strategy. Preliminary gating strategy used for the isolation of LSECs and KCs and the identification of HSCs, neutrophils, eosinophils, and monocytes. Representative gating scheme to identify cell populations within the liver of rAAV-transduced (A) control mice and (B) DT-treated mice 48 hours after DT injection. (A, B) Data are representative of two combined experiments (n = 4-6 per group). DT, diphtheria toxin and HSCs, hepatic stellate cells.

Supporting Figure 4. Depletion of monocytes does not alter peak liver injury. (A) Two weeks following rAAV injection, C57BL/6J mice received PBS and IgG2b isotype control antibody, DT and IgG2b isotype control antibody, or DT and anti-CCR2 antibody. Monocytes were depleted 48 hours following DT administration as shown by (B) representative flow plots and (C) monocyte numbers, while (E) neutrophils were not

depleted. Each data point represents an individual mouse (n = 5-8 per group), bars represent the median. Significance determined by three pairwise Mann-Whitney tests and the p-values adjusted for multiple comparisons using the Holm-Bonferroni method as described in the methods. *, P ≤ 0.05; **, P ≤ 0.01; ns, not significant. Ab, antibody; and DT, diphtheria toxin.

Supporting Figure 5. Depletion of neutrophils does not alter peak liver injury. (A) Two weeks following rAAV injection, C57BL/6J mice received PBS and IgG2a isotype control antibody, DT and IgG2a isotype control antibody, or DT and anti-Ly6G. Neutrophils were depleted 48 hours following DT administration as shown by (B) representative flow plots and (C) neutrophil numbers, while (E) monocytes were not depleted. Data are combined for 2 independent experiments. Each data point represents an individual mouse (n = 6 per group), bars represent the median. Significance determined by three pairwise Mann-Whitney tests and the p-values adjusted for multiple comparisons using the Holm-Bonferroni method as described in the methods. **, P ≤ 0.01; ns, not significant. Ab, antibody; and DT, diphtheria toxin.

Supporting Figure 6. Hepatocyte death induces expression of the TRIF pathway cytokine IFN-β while only moderately inducing expression of MyD88 pathway cytokines. mRNA expression relative to *Hprt* in total liver for *Ifnb1*, *Tnf*, *Il6*, *Il1a*, *Il1b*, and *Il12b* following DT injection of rAAV-transduced C57BL/6J mice. Data are combined from two independent experiments, each data point represents an individual mouse (n = 4-6 per group), bars represent the median. Significance determined by a Kruskal-Wallis test followed by Dunn's post-test of each time point to the No DT group. * represents

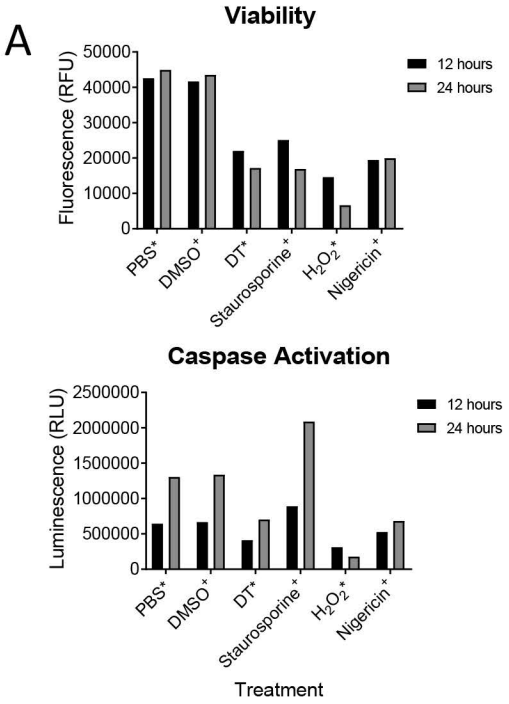
significance compared to No DT group; *, P ≤ 0.05; **, P ≤ 0.01; non-significance represented by absence of asterisk. DT, diphtheria toxin and ND, non-detect.

Supporting Figure 7. DT-induced death elicits similar gene expression profiles in Huh7 cells and mice. (A) Western blot for hTLR3 and β-actin in Huh7 and Huh7-TLR3 cells. (B) Fold change gene expression in Huh7 cells treated for 48 hours with 1, and 100 ng/mL DT compared to cells treated with only 1X PBS, *Hprt* used as the reference gene. Each bar represents two pooled wells run as a single sample. DT, diphtheria toxin.

Supporting Figure 8. Enrichment of cell-specific genes in hepatocyte, LSEC, and KC populations. Genes specific for hepatocytes, LSECs, KCs, and HSCs were measured in total liver, hepatocyte, LSEC, and KC RNA samples by qRT-PCR and expressed relative to *Hprt*. (A) Data combined from the two C57BL/6J time course experiments. All time points combined into one group per population. Each data point represents an individual mouse (n = 36-40 per group), bars represent the median, non-detects represented as zero. (B) Data are from 2 of 3 of the experiments performed in C57BL/6J and TRIF$^{-/-}$ mice. All four treatment groups combined into one group per population, each data point represents an individual mouse (n = 31-32 per group), bars represent the median, non-detects represented as zero. Hepa, hepatocyte; HSC, hepatic stellate cell; KC, Kupffer cell; and LSEC, liver sinusoidal endothelial cell.

References for Supporting Materials

1. Mack M, Cihak J, Simonis C, Luckow B, Proudfoot AEI, Plachý J, Brühl H, Frink M, et al. Expression and Characterization of the Chemokine Receptors CCR2 and CCR5 in Mice. J Immunol 2001;166:4697-4704.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12 hours
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H2O2*
Nigericin+
Caspase Activation
Luminescence (RLU)
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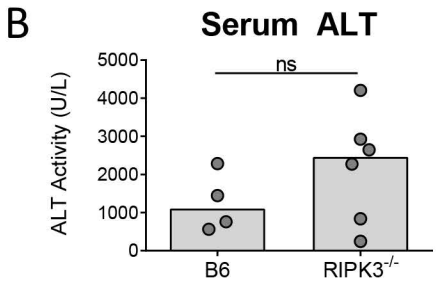
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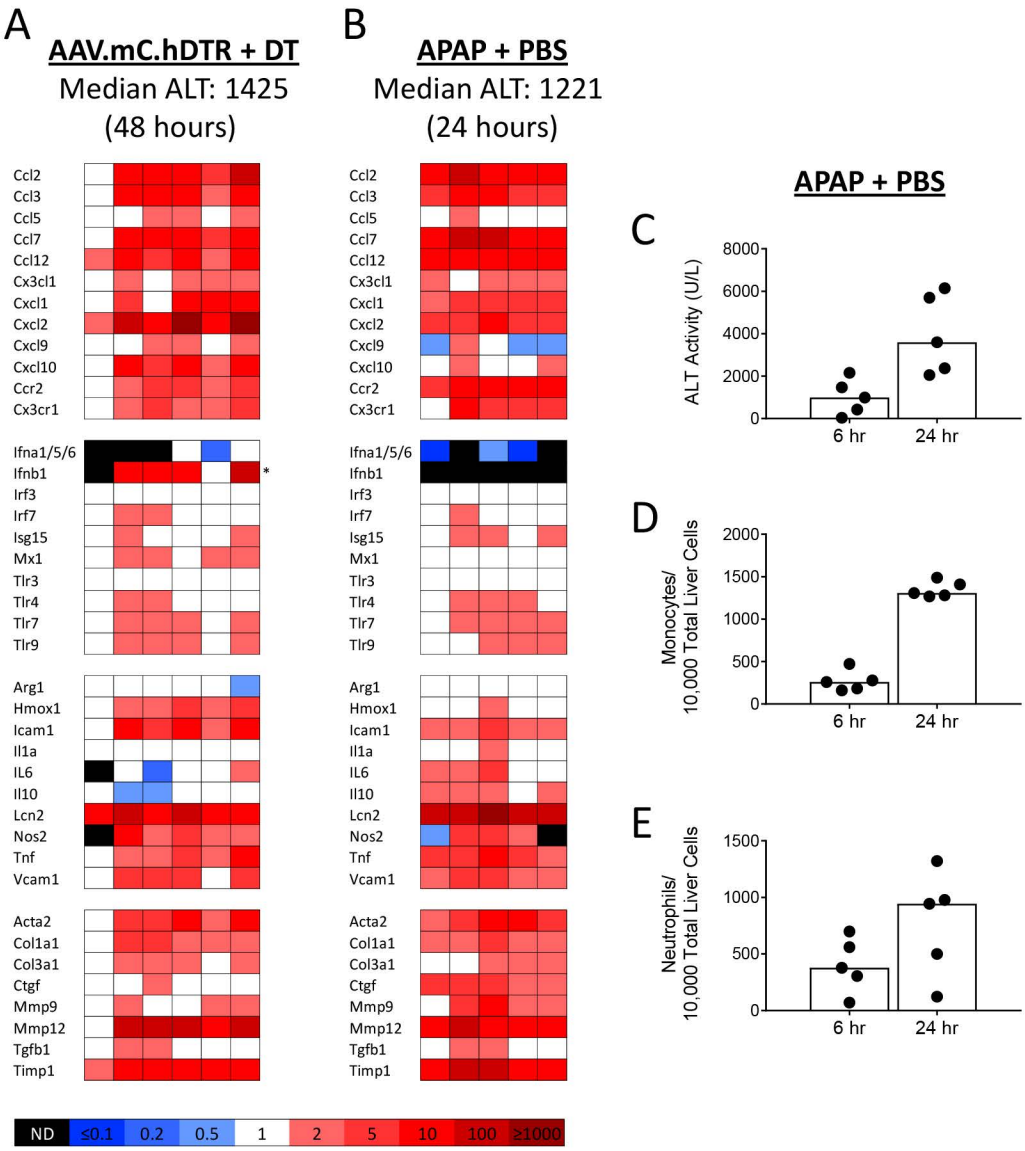
ns
B6
RIPK3-/-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(24 hours)
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Ccl5
Ccl7
Ccl12
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Ifnb1
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Irf7
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2
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≥1000
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ALT Activity (U/L)
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4000
2000
0
6 hr
24 hr
D
Monocytes/
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2000
1500
1000
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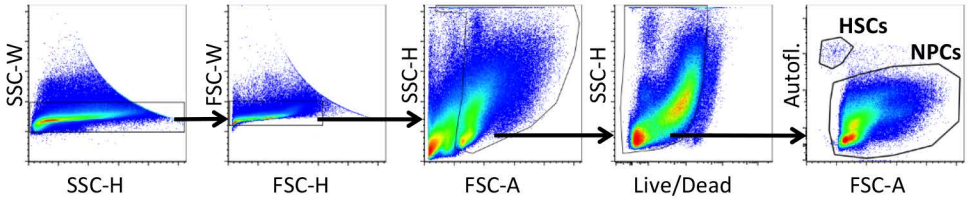
24 hr

A No diphtheria toxin



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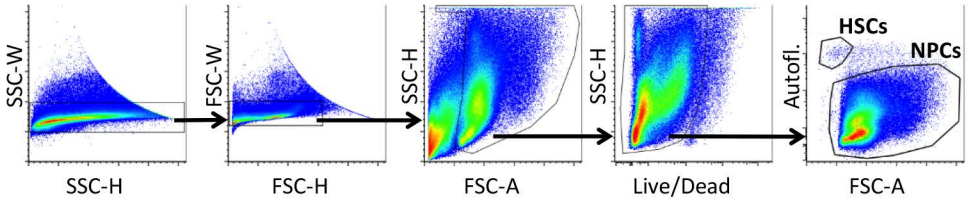

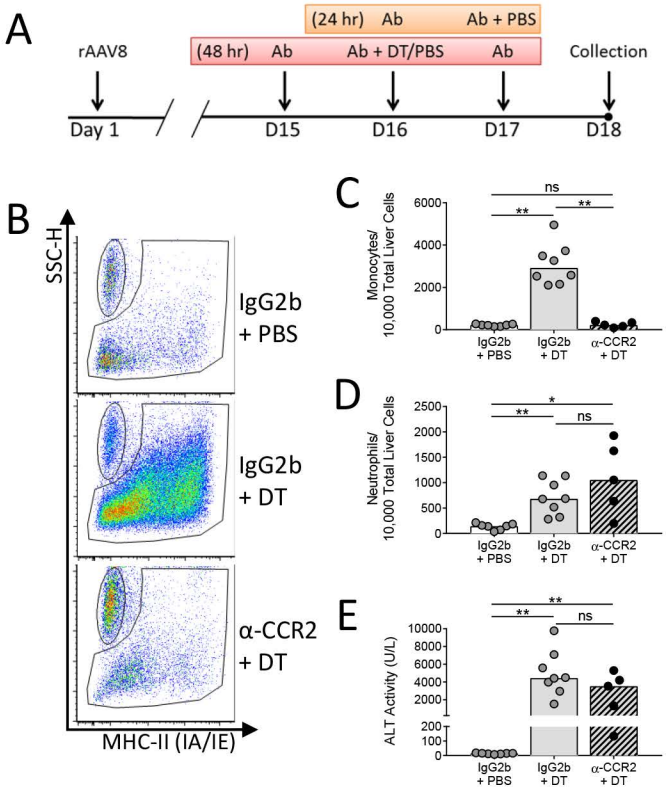
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+ PBS
IgG2b
+ DT
α-CCR2
+ DT
MHC-II (IA/IE)
C
Monocytes/
10,000 Total Liver Cells
ns
**
**
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4000
2000
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+ PBS
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+ DT
α-CCR2
+ DT
D
Neutrophils/
10,000 Total Liver Cells
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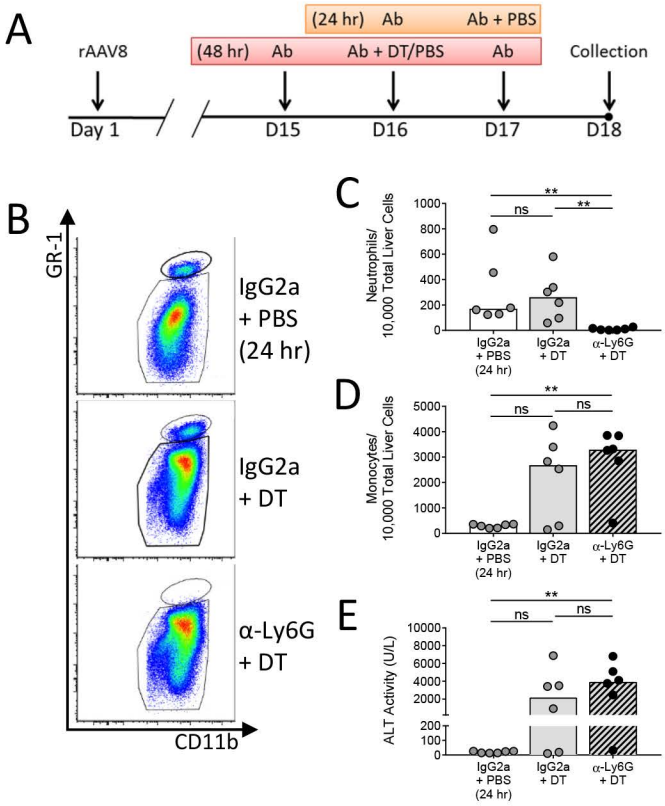
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IgG2a + DT
α-Ly6G + DT
C
Neutrophils/ 10,000 Total Liver Cells
D
Monocytes/ 10,000 Total Liver Cells
E
ALT Activity (U/L)
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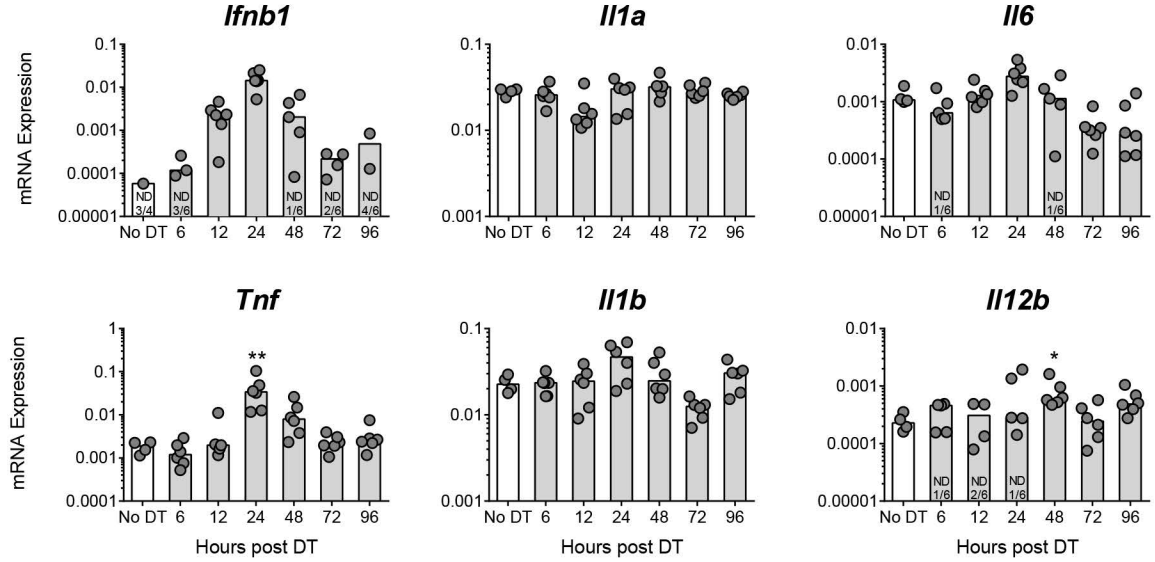
IgG2a + DT
α-Ly6G + DT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ND 1/6
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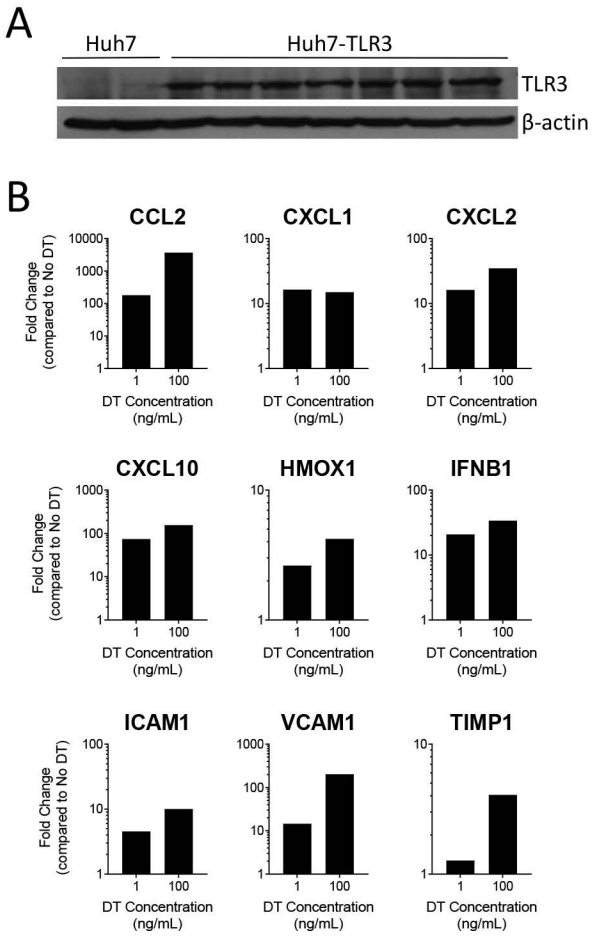
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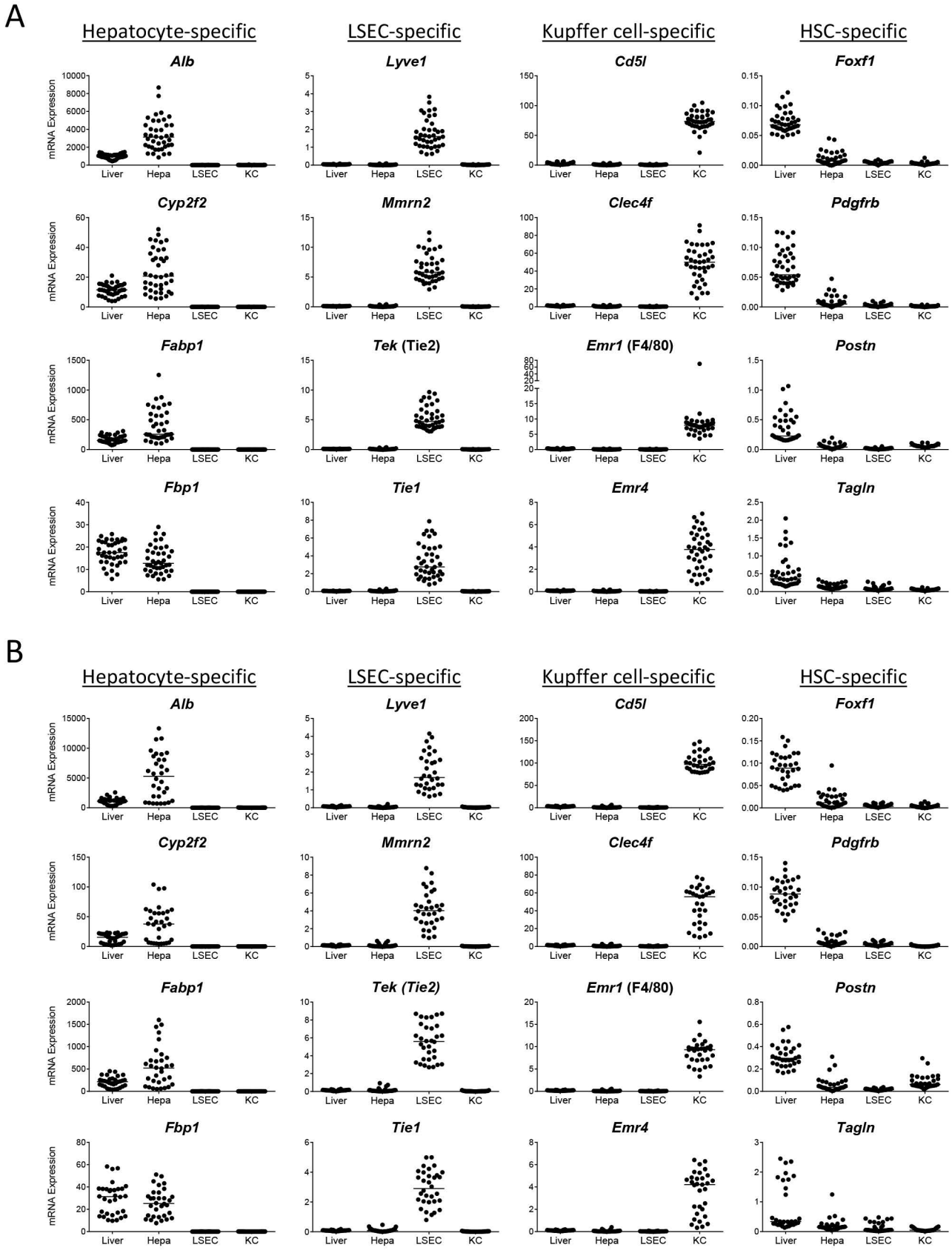
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Liver
Hepa
LSEC
KC
B
Hepatocyte-specific
LSEC-specific
Kupffer cell-specific
HSC-specific
Alb
Lyve1
Cd5l
Foxf1
Cyp2f2
Mmrn2
Clec4f
Pdgfrb
Fabp1
Tek (Tie2)
Emr1 (F4/80)
Postn
Fbp1
Tie1
Emr4
Tagln
mRNA Expression
Liver
Hepa
LSEC
KC

Gene	Assay ID	Species
Ccl2	Mm00441242_m1	Mouse
Ccl3	Mm00441259_g1	Mouse
Acta2	Mm00725412_s1	Mouse
Arg1	Mm00475988_m1	Mouse
Ccl12	Mm01617100_m1	Mouse
Ccl5	Mm01302427_m1	Mouse
Ccl7	Mm00443113_m1	Mouse
Ccr2	Mm00438270_m1	Mouse
Col1a1	Mm00801666_g1	Mouse
Col3a1	Mm01254476_m1	Mouse
Ctgf	Mm01192933_g1	Mouse
Cx3cl1	Mm00436454_m1	Mouse
Cx3cr1	Mm02620111_s1	Mouse
Cxcl1	Mm04207460_m1	Mouse
Cxcl10	Mm00445235_m1	Mouse
Cxcl2	Mm00436450_m1	Mouse
Cxcl9	Mm00434946_m1	Mouse

Hmox1	Mm00516005_m1	Mouse
Icam1	Mm00516023_m1	Mouse
Ifna1;Ifna6;Ifna5	Mm03030145_gH	Mouse
Ifnb1	Mm00439552_s1	Mouse
Il10	Mm00439614_m1	Mouse
Il12b	Mm00434174_m1	Mouse
Il1a	Mm00439620_m1	Mouse
Il1b	Mm00434228_m1	Mouse
Il6	Mm00446190_m1	Mouse
Irf3	Mm00516784_m1	Mouse
Irf7	Mm00516791_g1	Mouse
Isg15	Mm01705338_s1	Mouse
Lcn2	Mm01324470_m1	Mouse
Mmp12	Mm00500554_m1	Mouse
Mmp9	Mm00442991_m1	Mouse
Mx1	Mm00487796_m1	Mouse
Nos2	Mm00440502_m1	Mouse
Tgfb1	Mm01178820_m1	Mouse

Timp1	Mm00441818_m1	Mouse
Tlr3	Mm01207404_m1	Mouse
Tlr4	Mm00445273_m1	Mouse
Tlr7	Mm00446590_m1	Mouse
Tlr9	Mm00446193_m1	Mouse
Tnf	Mm00443258_m1	Mouse
Vcam1	Mm01320970_m1	Mouse
CCL2	Hs00234140_m1	Human
CXCL1	Hs00605382_gH	Human
CXCL10	Hs01124252_g1	Human
CXCL2	Hs00601975_m1	Human
HMOX1	Hs01110250_m1	Human
HPRT1	Hs02800695_m1	Human
ICAM1	Hs00164932_m1	Human
IFNB1	Hs01077958_s1	Human
TIMP1	Hs00171558_m1	Human
VCAM1	Hs01003372_m1	Human

Supporting Table 1. TaqMan Assays.