Supplementary Table and Figure Legends

Supplementary Table S1 siRNA for lncRNA screening

(A) Expression of lncRNAs for functional screening. RPKMs (reads per kilobase per million mapped reads) were calculated from RNA-seq data from LPS-treated BMDMs.

(B) Shown are sequences of siRNA that were used for functional screening of lncRNAs.

Supplementary Table S2 GSEA analysis of the effects of *lncFAO* knockdown

BMDMs transfected with anti-*lncFAO* (silncFAO #1) or control siRNA as indicated in Fig. 1A were treated with LPS. Twenty-four hours after the LPS treatment, the cells were subjected to polyA RNA-seq, and the RNA-seq data were analyzed using GSEA.

Supplementary Table S3 GSEA analysis of *lncFAO*$^{-/-}$ BMDMs activated with LPS

BMDMs from WT and *lncFAO*$^{-/-}$ (KO1) mice were treated with LPS, and RNA was analyzed at the indicated time points using RNA-seq. Shown are FDR < 0.05 MSigDB hallmark gene sets. The gene sets identified at 4 or 5 time points are shown in red. n=3 for each group at each time point.

Supplementary Table S4 Resolution/repair phase Ly6C^{hi} macrophage signature genes

Genes in the gene sets identified in Fig. 2E are shown.

Supplementary Figure S1 Characterization of *lncFAO*

(A) To screen the function of newly identified lncRNAs, eleven lncRNAs whose expression

we detected in LPS-treated BMDMs were knocked down using specific siRNAs. RNAs were obtained 24 h after LPS treatment, and *Il1a* expression was analyzed using qPCR. **P*<0.05 vs. control siRNA-transfected cells. Tukey-Kramer's post-hoc test.

(B) siRNA-mediated *lncFAO* knockdown. Levels of *lncFAO* mRNA were analyzed in the RNA analyzed in Fig. 1a. mRNA levels were first normalized to those of 18s rRNA and then to the level in untreated control siRNA-transfected BMDMs. n=3. **P*<0.05 vs. control siRNA at the same time point. Two-tailed unpaired Student's *t* test.

(C) RNA was purified from the nuclear and cytoplasmic fractions. Shown are relative *lncFAO* transcript levels in the two fractions. n=3. Two-tailed unpaired Student's *t* test.

(D) Levels of *lncFAO* mRNA in BMDMs and tissues collected from mice in a steady-state. mRNA levels were first normalized to those of 18s rRNA and then to the level in the untreated BMDMs. n=3 for each group. ND, not detected.

(E) Levels of cytokine gene expression in macrophages in myocardial infarction described in Fig. 1D.

Supplementary Figure S2 Single-cell RNA-seq analysis of *lncFAO* after myocardial infarction

(A) Single-cell RNA-seq data sets (ArrayExpress E-MTAB-7376) (1) were integrated using Seurat ver. 3 (2). Expression levels of *lncFAO* and lineage marker genes in 35,192 cells are shown in a t-SNE plot. Most *lncFAO*-expressing cells were found in $Ptprc^{+}Itgam^{+}Cd68^{+}$ populations.

(B, C) Cell populations expressing *Ptprc*, *Itgam* and *Cd68* were analyzed further. Clustering of the cells and analysis of marker and signature genes revealed 8 subpopulations.

Ly6c2^{hi}*Ccr2*^{hi}*Adgre1*^{int} cells (cluster 1) appear to correspond to Ly6C^{hi}F4/80^{lo} macrophages (3). *Ly6c2*^{hi}*Ccr2*^{hi}*Adgre1*^{int} and two populations of *Ly6c2*^{int}*Msr1*^{hi}*Arg1*$^{+}$ cells (cluster 2, 3) expressed *Ccr2* and accumulated 3 days post-MI, suggesting they are monocyte-derived cells recruited through CCR2/CCL2 signaling. *Ly6c2*^{lo}*Adgre1*^{hi}*Cx3cr1*^{hi} cells (cluster 4, 5) likely include tissue-resident macrophages in sham hearts (4, 5). After MI, however, these populations also contained *Ccr2*$^{+}$ cells, suggesting they include monocyte-derived cells acquiring a tissue-resident macrophage-like phenotype (6). *Ptprc*$^{+}$*Itgam*$^{+}$*Cd68*$^{+}$ cells also contained minor population of *Lgals3*$^{+}$*Fabp5*$^{+}$ macrophages (cluster 6), *Itgax*$^{+}$*Cd209a*$^{+}$*Adgre1*$^{-}$ classical dendritic cells (DCs) (cluster 7), and *Ly6c2*^{lo}*Cd64*$^{-/lo}$*Cd68*^{lo}*Nr4a1*$^{+}$ monocytes (cluster 8), which likely correspond to Ly6C^{lo} patrolling monocytes.

Supplementary Figure S3 Characterization of *lncFAO*-expressing subpopulations of macrophages after myocardial infarction

(A) Expression of macrophage markers and pro- and anti-inflammatory cytokines in the clusters of cells identified in Supplementary Fig. S2 in Sham-operated and day 3 and day 7 post-MI hearts.

(B) Gene ontology enrichment analysis of the more highly expressed genes in *lncFAO*-expressing clusters on day 7 (Cluster 1, 378 genes; Cluster 2, 71 genes). Shown are gene sets with $q<10^{-5}$.

(C) GSEA analysis of the genes differentially expressed in a cluster in comparison to the remaining clusters on day 7. Shown are gene sets with $q<0.1$.

Supplementary Figure S4 Generation of *lncFAO*$^{-/-}$ mice

(A) Positions of single guided RNAs (sgRNAs), the deleted region, and the primers for genomic PCR analysis of a *lncFAO*$^{-/-}$ line (KO1) are shown schematically. The deleted region is shown as a dotted line.

(B) Genomic PCR of the targeted region in wild-type (WT) mice and in two lines of *lncFAO*$^{-/-}$ (KO1 and KO2) mice. The targeted WT allele generated the 950 bp band as depicted in a.

(C) Analysis of the off-target mutations. PCR was performed to amplify three off-target candidate sites, which were predicted by CRISPR Design (http://crispr.mit.edu/). After denaturing and reannealing the amplified DNA fragments, they were incubated with Guide-it resolvase (Takara), which recognizes mismatches and cleaves dsDNA at the mismatched sites. The PCR products for off-target sites did not generate cleaved fragments. The amplified DNA fragment of the target region was used as a positive control. Shown are results from the KO1 line.

(D) Body weight changes in male WT and *lncFAO*$^{-/-}$ (KO1) mice. There were no significant differences between the two groups at the indicated ages. n=6 for each group. Shown are means ± S.D. Data were analyzed using unpaired Student's *t* test at each age. NS, not significant.

(E) *lncFAO* expression in BMDMs from WT mice and two lines of *lncFAO*$^{-/-}$ (KO1 and KO2) mice after LPS treatment. mRNA levels were first normalized to those of 18s rRNA and then to the level in untreated WT BMDMs. n=3. Tukey-Kramer's post-hoc test.

(F) qPCR analysis of *Il6* in BMDMs from WT and the KO2 line of *lncFAO*$^{-/-}$ mice. mRNA levels were first normalized to those of 18s rRNA and then to the level in untreated (0 h) WT BMDMs. n=3 for each group. *P<0.05 between, two-tailed unpaired Student's *t*-test.

Supplementary Figure S5 Nuclear translocation of NF-κB in LPS-treated BMDMs

Representative micrographs showing immunofluorescent staining of NF-κB p65 (red) in BMDMs treated with LPS for the indicated times. Nuclei were counterstained with DAPI (green). Scale bars, 10 μm.

Supplementary Figure S6 Effects of *lncFAO* deletion on skin wound healing

(A) Representative skin wounds 8 days after a skin excision injury.

(B) Representative histological images of skin wounds on day 8 post-injury. Masson's Trichrome staining of sections from mice transplanted with WT or *lncFAO*$^{-/-}$ bone marrow. Scale bars, 500 μm.

Supplementary Figure S7 Identification of *lncFAO* binding proteins

(A) Image of a silver-stained SDS-PAGE gel. Whole cell lysates of BMDMs left untreated for 24 h or 24 h after LPS treatment were incubated with *in vitro*-transcribed biotinylated *lncFAO* RNA, after which the RNA and binding proteins were pulled down using streptavidin-coated Dynabeads (lanes 2 and 4). As negative controls, lysates of LPS-treated BMDMs were incubated with biotinylated antisense RNA (lane 5) or without biotinylated RNA (lane 3). The band in the orange square was extracted for LC-MS/MS analysis. Lane 1 shows size markers.

(B) Proteins in the excised band indicated in A and detected using LC-MS/MS.

Supplementary Figure S8 Pull-down of endogenous *lncFAO* complexes

Relative abundances of the target and non-target RNAs in samples pulled down with anti-

lncFAO oligos as compared to the abundances in the input whole cell lysates. The purified samples and the input whole cell lysates were reverse transcribed, and the abundances of the indicated RNA species were analyzed by qPCR. The results show a specific enrichment of *lncFAO* in the pull down.

Supplementary Figure S9 lncFAO localization in LPS-treated BMDMs

lncFAO RNA and mitochondrial COX-IV protein were visualized using fluorescent in-situ hybridization (FISH) and immunofluorescent staining in WT-BMDMs left untreated (A) or treated for 24 h with LPS (B). In C, *lncFAO*$^{-/-}$ BMDMs were stained as a negative control. Scale bars, 10 μm.

Supplementary Figure S10 *Hadhb* knockdown

Quantitative PCR analysis of *Hadhb* expression in BMDMs transfected with control (siControl) or siRNA targeting *Hadhb* or *lncFAO* as described in Figure 4F (A) and 4G (B). mRNA levels were first normalized to those of 18s rRNA and then to the level in siControl-transfected BMDMs. n=3. In A, **P*<0.05 vs. siControl. Tukey-Kramer's post-hoc test. In B, **P*<0.05 vs. siControl at the same time point. Two-tailed unpaired Student's *t* test.

Supplementary Figure S11 *Hadhb* knockdown in *lncFAO*$^{-/-}$ BMDMs

BMDMs from WT and *lncFAO*$^{-/-}$ mice were transfected with siRNA targeting *Hadhb* or control siRNA. Twenty-four hours after transfection, the cells were treated with LPS.

(A) IL-6 levels in the medium were analyzed 24 h after LPS treatment of WT and *lncFAO*$^{-/-}$ BMDMs. n=3. *P<0.05 vs. siControl of the same genotype, $^{\#}P$<0.05 vs WT of the same siRNA treatment, Tukey-Kramer's post-hoc test.

(B) Quantitative PCR analysis of *Hadhb* expression in BMDMs as described in A. *P<0.05 vs. siControl of the same genotype, Tukey-Kramer's post-hoc test.

Refernces

1. Farbehi N, *et al.* (2019) Single-cell expression profiling reveals dynamic flux of cardiac stromal, vascular and immune cells in health and injury. *eLife* 8:e43882.
2. Stuart T, *et al.* (2019) Comprehensive Integration of Single-Cell Data. *Cell* 177(7):1888-1902.e1821.
3. Fujiu K, *et al.* (2017) A heart-brain-kidney network controls adaptation to cardiac stress through tissue macrophage activation. *Nat. Med.* 23(5):611-622.
4. Epelman S, *et al.* (2014) Embryonic and Adult-Derived Resident Cardiac Macrophages Are Maintained through Distinct Mechanisms at Steady State and during Inflammation. *Immunity* 40(1):91-104.
5. Swirski FK & Nahrendorf M (2018) Cardioimmunology: the immune system in cardiac homeostasis and disease. *Nature Reviews Immunology* 18(12):733-744.
6. Dick SA, *et al.* (2019) Self-renewing resident cardiac macrophages limit adverse remodeling following myocardial infarction. *Nat. Immunol.* 20(1):29-39.

Refernces

1. Farbehi N, *et al.* (2019) Single-cell expression profiling reveals dynamic flux of cardiac stromal, vascular and immune cells in health and injury. *eLife* 8:e43882.
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4. Epelman S, *et al.* (2014) Embryonic and Adult-Derived Resident Cardiac Macrophages Are Maintained through Distinct Mechanisms at Steady State and during Inflammation. *Immunity* 40(1):91-104.
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6. Dick SA, *et al.* (2019) Self-renewing resident cardiac macrophages limit adverse remodeling following myocardial infarction. *Nat. Immunol.* 20(1):29-39.

A

Gene name	Gene locus (mm9)	RPKM Baseline	LPS 4h	24h	48h
LincRNA_LPS2	chr10: 66,374,941 - 66,381,731	0.3	0.9	2.4	0.8
LincRNA_LPS4	chr10: 44,315,002 - 44,429,890	0.2	0.3	0.4	0.9
LincRNA_LPS5	chr16: 3,754,741 - 3,763,969	0.0	0.1	0.1	0.0
LincRNA_LPS6	chr6: 86,619,982 - 86,621,267	0.1	0.2	0.1	0.6
LincRNA_LPS8	chr16: 93,128,061 - 93,142,507	0.0	0.4	3.1	0.3
LincRNA_LPS9	chr10: 87,158,901 - 87,159,185	0.0	0.0	1.9	2.6
LincRNA_LPS11	chr11: 117,839,170 - 117,840,370	0.0	0.9	0.4	0.1
LincRNA_LPS12	chr11: 97,523,72 - 97,524,435	0.1	2.4	1.8	1.0
LincRNA_LPS18	chr17: 29,626,262 - 29,627,368	2.0	1.2	2.7	2.9
LincRNA_LPS19	chr19: 12,598,652 - 12,605,701	0.0	26.3	13.2	4.3
lncFAO	chr13: 52,649,726 - 52,668,660	3.9	0.9	12.7	6.1

B

Gene name	Sense	Antisense
LincRNA_LPS2	GCGAAGGAGAGAGAGAGAAAUCCUU	GCGAAGGAGAGAGAGAGAAATCCTT
LincRNA_LPS4	UGUGCCCUAUGGAAGAUCUGGAUUA	TGTGCCCTATGGAAGATCTGGATTA
LincRNA_LPS5	CAGUGAAGGGAAGAAGGAAUGUGCA	CAGTGAAGGGAAGAAGGAATGTGCA
LincRNA_LPS6	GGGCAUGCCUUGAUGUGAACCUUUA	GGGCATGCCTTGATGTGAACCTTTA
LincRNA_LPS8	AGGAGGCAAUAGAGCGGCAUCUAAU	AGGAGGCAATAGAGCGGCATCTAAT
LincRNA_LPS9	GAAGGCCAGAUACACUCCAUCUUCA	GAAGGCCAGATACACTCCATCTTCA
LincRNA_LPS11	CCCAGAAUUCUAGAAGGCCUCUCUA	UAGAGAGGCCUUCUAGAAUUCUGGG
LincRNA_LPS12	GAGCUGCUUUGAAACUUCUUACCAA	UUGGUAAGAAGUUUCAAAGCAGCUC
LincRNA_LPS18	CAUCGUCUUAUAGUCCUCAUAGUUU	AAACUAUGAGGACUAUAAGACGAUG
LincRNA_LPS19	UCUGCCCGGCUAACACCCAUUUCUU	TCTGCCCGGCTAACACCCATTTCTT

Supplementary Table S1

Gene set	NES	FDR q-val
HYPOXIA	2.3624	0.0000
TNFA_SIGNALING_VIA_NFKB	2.3437	0.0000
INFLAMMATORY_RESPONSE	2.2399	0.0000
TGF_BETA_SIGNALING	2.0193	0.0007
KRAS_SIGNALING_UP	1.9961	0.0006
IL6_JAK_STAT3_SIGNALING	1.9884	0.0006
COMPLEMENT	1.9743	0.0006
INTERFERON_GAMMA_RESPONSE	1.9606	0.0005
EPITHELIAL_MESENCHYMAL_TRANSITION	1.9469	0.0005
XENOBIOTIC_METABOLISM	1.8900	0.0013
ALLOGRAFT_REJECTION	1.8679	0.0016
P53_PATHWAY	1.8330	0.0027
INTERFERON_ALPHA_RESPONSE	1.8045	0.0034
IL2_STAT5_SIGNALING	1.7577	0.0057
APOPTOSIS	1.7382	0.0066
COAGULATION	1.7301	0.0067
REACTIVE_OXIGEN_SPECIES_PATHWAY	1.6417	0.0148
UV_RESPONSE_DN	1.6287	0.0157
HEME_METABOLISM	1.6274	0.0151
GLYCOLYSIS	1.5341	0.0351
MTORC1_SIGNALING	1.4977	0.0455
ESTROGEN_RESPONSE_EARLY	1.4911	0.0460

Supplementary Table S2

Upregluated in *lncFAO* $^{-/-}$

0 h

Gene set	NES	FDR q
Myc targets v1	2.4	0
Myc targets v2	2.4	0
Unfolded protein response	2.14	0
Angiogenesis	2.11	0
Tgf beta signaling	2.09	0
Tnfa signaling via NFkB	1.92	0.001
Mtorc1 signaling	1.89	0.001
Wnt beta catenin signaling	1.84	0.002
Notch signaling	1.65	0.016
Interferon alpha response	1.63	0.017
Cholesterol homeostasis	1.62	0.017
G2M checkpoint	1.61	0.016
E2F targets	1.55	0.027
Inflammatory response	1.49	0.038
Estrogen response early	1.48	0.037
UV response up	1.44	0.049

4 h

Gene set	NES	FDR q
Interferon alpha response	2.57	0
Myc targets v1	2.47	0
Interferon gamma response	2.42	0
Tnfa signaling via NFkB	2.34	0
Inflammatory response	2.07	0
MTORC1 signaling	2.06	0
Unfolded protein response	2.02	0
Allograft rejection	2.01	0
UV response up	1.76	0.003
Wnt beta catenin signaling	1.73	0.004
IL2 STAT5 signaling	1.69	0.006
Myc targets v2	1.67	0.007
Oxidative phosphorylation	1.65	0.008
Complement	1.6	0.013
Notch signaling	1.48	0.039
IL6 JAK STAT3 signaling	1.45	0.049

8 h

Gene set	NES	FDR q
Inflammatory response	2.03	0
Tnfa signaling via NFkB	2	0
Myc targets v1	1.93	0
Myc targets v2	1.89	0.002
Allograft rejection	1.84	0.004
Unfolded protein response	1.82	0.003
IL2 STAT5 signaling	1.65	0.018
Mitotic spindle	1.65	0.016

12 h

Gene set	NES	FDR q
Inflammatory response	2.26	0
Interferon gamma response	2.26	0
Interferon alpha response	2.24	0
Allograft rejection	2.14	0
Tnfa signaling via NFkB	2.02	0.001
Tgf beta signaling	1.78	0.02
Mitotic spindle	1.78	0.017
Wnt beta catenin signaling	1.65	0.044

24 h

Gene set	NES	FDR q
Interferon gamma response	2.29	0
Interferon alpha response	2.22	0
Inflammatory response	2.18	0
Allograft rejection	1.96	0.001

Downregluated in *lncFAO* $^{-/-}$

0 h

Gene set	NES	FDR q
Bile acid metabolism	-0.6	0.002

4 h

Gene set	NES	FDR q
None		

8 h

Gene set	NES	FDR q
Hypoxia	-1.73	0.042
Xenobiotic metabolism	-1.68	0.037
Heme metabolism	-1.64	0.039
Glycolysis	-1.63	0.031

12 h

Gene set	NES	FDR q
Hypoxia	-1.75	0.033

24 h

Gene set	NES	FDR q
None		

Supplementary Table S3

Cardiac *Ly6c2*^{hi} day 7 post-MI upregulated genes

Ly6a, Slamf9, Klf4, Cav1, Ctla2a, Sh2d1b1, Snhg12, Ccdc88c, Sema4d, Rnase6, Icam2, Cx3cr1, Rgs2, Rbks, Hhex, Stat1, Batf3, Ifi203, Spn, Epsti1, Grk3, Dusp2, Pttg1, Clec2i, Ace, Fh1, Myo1g, Cdc42ep3, Gsto1, Ly6c1, Pdlim1, Rasa4, Klf2, Sparcl1, Lmo4, Bmyc, Ckb, Gng11, Rasgrp2, Cbfa2t3, Irf1, Ramp1, Hscb, Hmgb2, H1f0, Hspb1, Lpl, Nfe2, Cd81, Susd3, Rbfa, Nsmce4a, Cd74, Igfbp7, Col3a1, Mef2c, Igbp1, Calhm2, Racgap1, Pmaip1, Tuba1a, Surf1, Tsc22d1, Gpr141, Slamf8, Mgp, Mbp, Dcn, Gimap6, Rogdi, Arl5c, Aw112010, Nedd9, Sparc, Rnf166, Arhgap15, Ogt, Rp9, Tifa, Tm4sf1, Fabp4, Cd36, Gstp1, Mettl7a1, Gpx3, Pou2f2, Chst12, Bgn, Tmcc1, Limd2, Arhgap17, Ciita, Ldlrad3, Cyp27a1, Ptpro, Fcgrt, Ppfia4, Hfe, Tmem176a, Ypel3, Pltp, Lyl1, Ccng2, Igfbp4, Il16, St8sia4, Hpgd

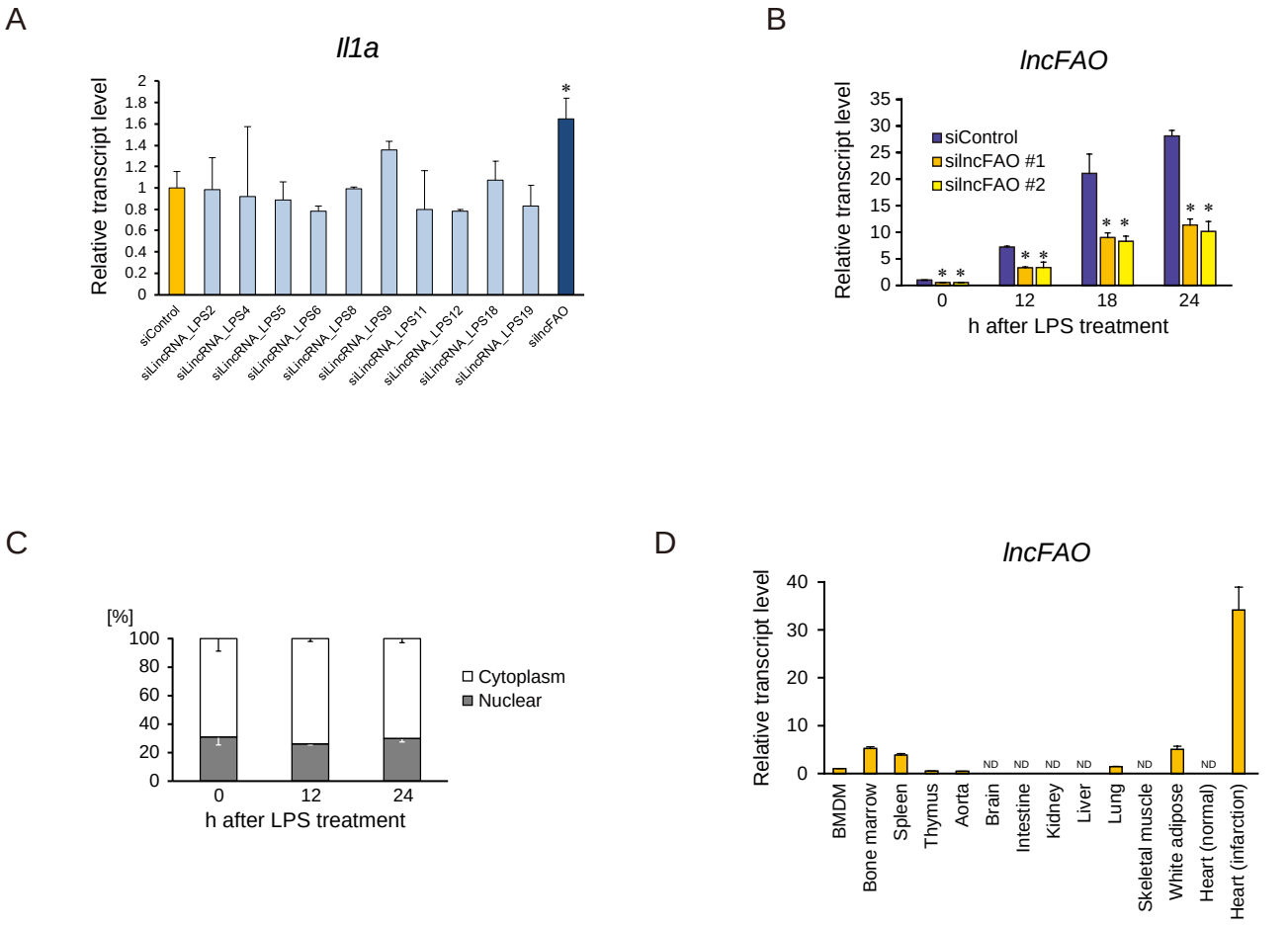
Cardiac *Ly6c2*^{hi} day 7 post-MI downregulated genes

Plaur, Slc39a14, Trem1, Ccl2, Cd300lf, Slc16a3, Ccl7, Eif1a, Ddx21, Chd7, Tubb6, Map2k3, Slc2a1, Fkbp5, Uck2, Furin, Sdc4, B4galt5, Adora2b, Hyou1, Spint1, Smox, Cd44, Plk3, Slpi, Snx18, Tgm2, Ehd1, Plec, Ccr1, Txnrd1, Rhov, Phlda1, C5ar1, Lilr4b, Rab7b, Srxn1, Hif1a, Card19, Lrrc59, Fam129b, Hmox1, Basp1, Dnaja1, Gpat3, Notch2, Cxcl2, Psmc1, Dok2, Arg1, Gcnt2, Manf, S100a11, Rab20, Ifi207, Rap2b, Nars, Slc7a11, Chil3, Adam8, Kdm6b, Creld2, Clec4e, Fcgr1, Nab2, Myh9, Wfdc21, Tarm1, Chordc1, Dmkn, Mt1, Sdf2l1, Ist1, Hnrnpm, Tuba1c, Slfn2, Ldha, Actb, Thbs1, Emilin2, Gk, Lcn2, Spp1, Lmnb1, Hp, S100a8, Cstb, Ctsl, Ms4a6d, Actr2, Rnh1, Tpm4, Msr1, Picalm, Slc15a3, Il1rn, Por, Vcan, Osm, Clec4d, Atp1a1, Hspa5, Mafb, Slc39a1, Cd14, Ctsd, Saa3, Ier3, Il4ra, Rrbp1, Tpd52, Alkbh5, Lrg1, Ctsb, Igfbp6, Hsp90b1, Ninj1, Bst1, Clec4n, Pdia6, Plek, Bcl2l11, F10, Itgam, Fbxl5, Eps8, S100a9, Srgn, Mdfic, Alas1, Myof, Stab1, Lgmn, Ccr5, Egr1, Ecm1, Gda, Mxd1, Wfdc17, Ptpn1, Mmp19, Maf, Ptafr, Mmp8

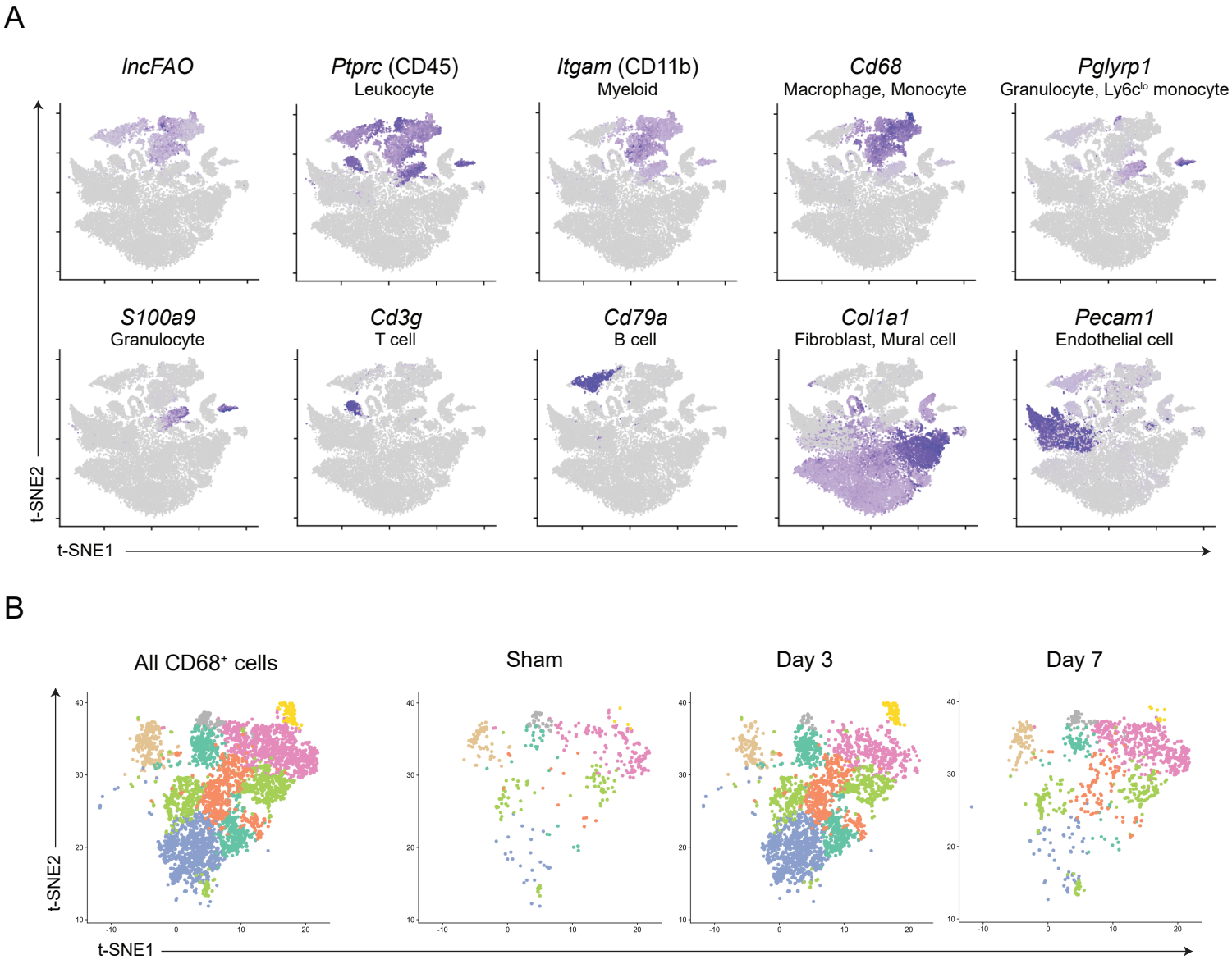
Skeletal muscle Ly6C^{hi} day 4 post-injury signature genes

Mcm2, Spsb1, Fads2, Pick1, Tbrg1, Atp6v0a2, Pigo, Mcm5, Lrrc40, Hmgn2, Cdca7l, Mcm7, Rab2b, Slfn9, Zdhhc24, Pmpca, Phax, Ppcdc, Dnase1l3, Wars2, Cbx5, Snx19, Psmd13, Rbl1, H2-t24, Aplnr, Znfx1, Chml, Recql5, Lclat1, Fadd, Dhdh, Chchd5, Fam122b, Nicn1, Mad2l1, Smarcd1, Slc46a1, Crbn, Cd99l2, Atp1b1, Cenpo, Acsm3, Pnpla6, Tmem229b, Pigb, Galns, Pmm2, Dctn5, Kif9, Klhl6, Tst, Nid2, Usp11, Tubb5, Zfp398, Mtmr4, Zkscan3, Ppip5k1, Rbbp9, Surf2, Kif18b, Il18bp, Spns3, Eri2, Pcp4l1, Asf1b, Uhmk1, Med22, Tbck, Pigh, Aldh7a1, Fbxo25, Rab11b, Fancg, Man2c1, Tbl1x, Mbd4, Surf1, Apod, Mfap3, Scd2, Plekhh1, Map3k12, Cnot6l, Gatc, Zhx1, Btbd2, Poc1b, Zc3h12d, Cd37, Trit1, Rad50, Glb1, Ccdc6, Leprot, Bscl2, Ift122, Xpc, Casp9, Plbd2, Adcy3, Oxa1l, Dars2, Map3k4, Siae, Ccni, Cdk5rap2, Znrf2, Mettl7a1, Abl1, Ube4b, Slc29a3, Chek2, Pld3, Crat, Naglu, Calcoco1, Ap3b1, Pdk2, Os9, Ezh1, Nbr1, Lmf2, Ccpg1, Plxnc1, Sft2d2

Supplementary Table S4

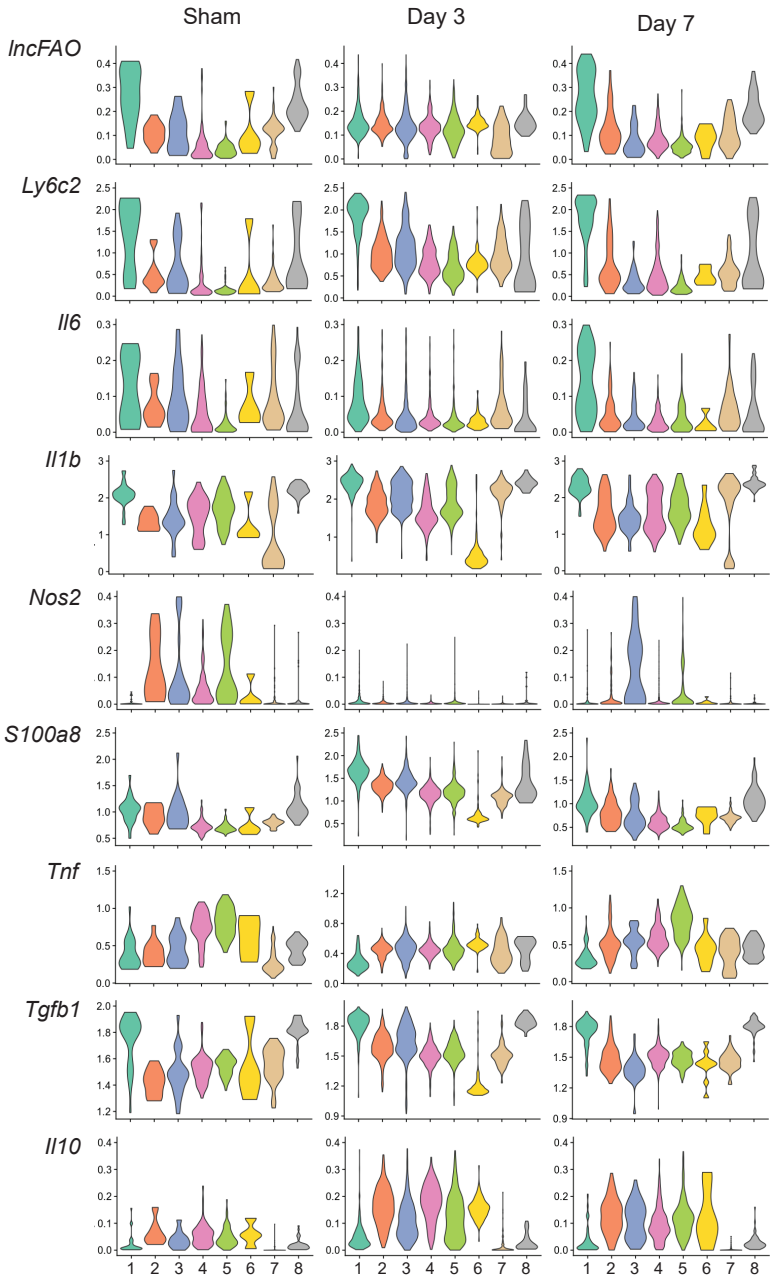


Supplementary Figure S1



Supplementary Figure S2

A



B

Cluster 1 at day 7

Term	Description	Log(q-value)
R-MMU-6798695	Neutrophil degranulation	-20.179
GO:0006954	Inflammatory response	-11.392
R-MMU-109582	Hemostasis	-10.798
GO:0097529	Myeloid leukocyte migration	-10.668
GO:0007159	Leukocyte cell-cell adhesion	-10.484
GO:0002683	Negative regulation of immune system process	-7.800
GO:0006909	Phagocytosis	-7.375
mmu04380	Osteoclast differentiation	-7.108
GO:0045123	Cellular extravasation	-6.225
GO:0048514	Blood vessel morphogenesis	-6.127
GO:0002695	Negative regulation of leukocyte activation	-5.763
GO:0070372	Regulation of ERK1 and ERK2 cascade	-5.479
GO:0072593	Reactive oxygen species metabolic process	-5.417
GO:0019221	Cytokine-mediated signaling pathway	-5.398
GO:0097190	Apoptotic signaling pathway	-5.398
GO:0002274	Myeloid leukocyte activation	-5.398
GO:0034341	Response to interferon-gamma	-5.208
GO:0042060	Wound healing	-5.050
GO:0051051	Negative regulation of transport	-5.050

Cluster 2 at day 7

Term	Description	Log(q-value)
GO:0097529	Myeloid leukocyte migration	-15.745
R-MMU-6798695	Neutrophil degranulation	-10.758
GO:0071674	Mononuclear cell migration	-7.763
GO:0001525	Angiogenesis	-6.652
GO:0042060	Wound healing	-6.476
GO:0030155	Regulation of cell adhesion	-5.931

C

Cluster 1 at day 7

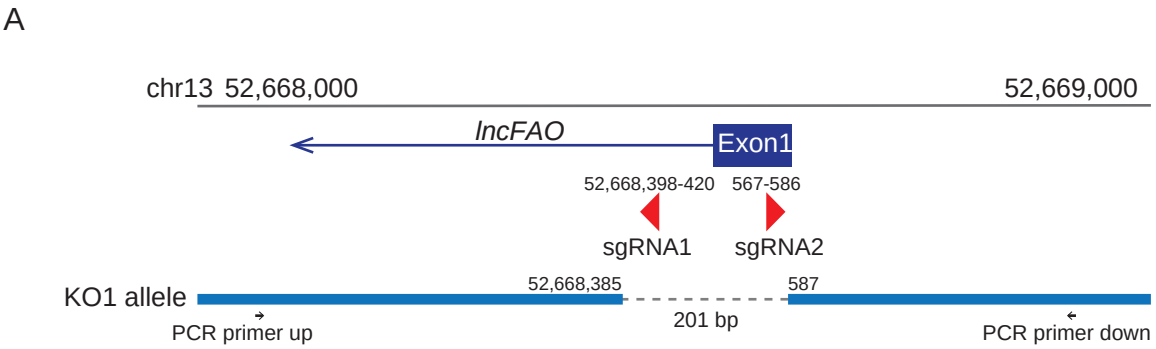
Term	NES
Oxidative phosphorylation	2.01
Reactive oxygen species pathway	1.93
Complement	1.86
Interferon gamma response	1.82
Allograft rejection	1.74
Cogagulation	1.65
Cholesterol homeostasis	1.63
Il2 stat5 signaling	1.59

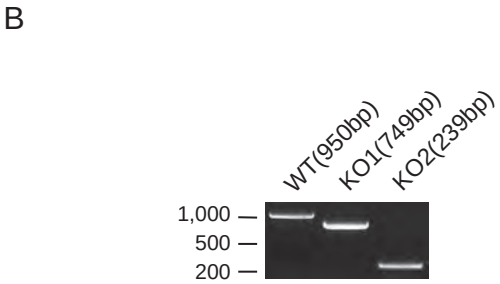
Cluster 2 at day 7

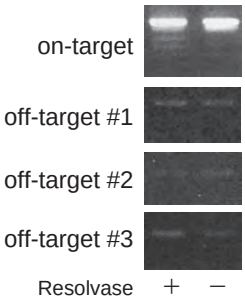
Term	NES
Mtorc1 signaling	1.85
Myc targets v1	1.63
Glycolysis	1.62

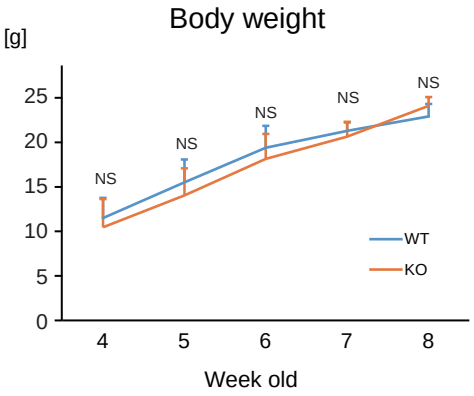
FDR <0.05
FDR >0.05, <0.1

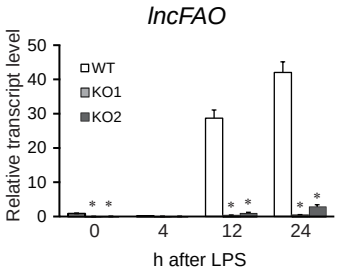
Supplementary Figure S3



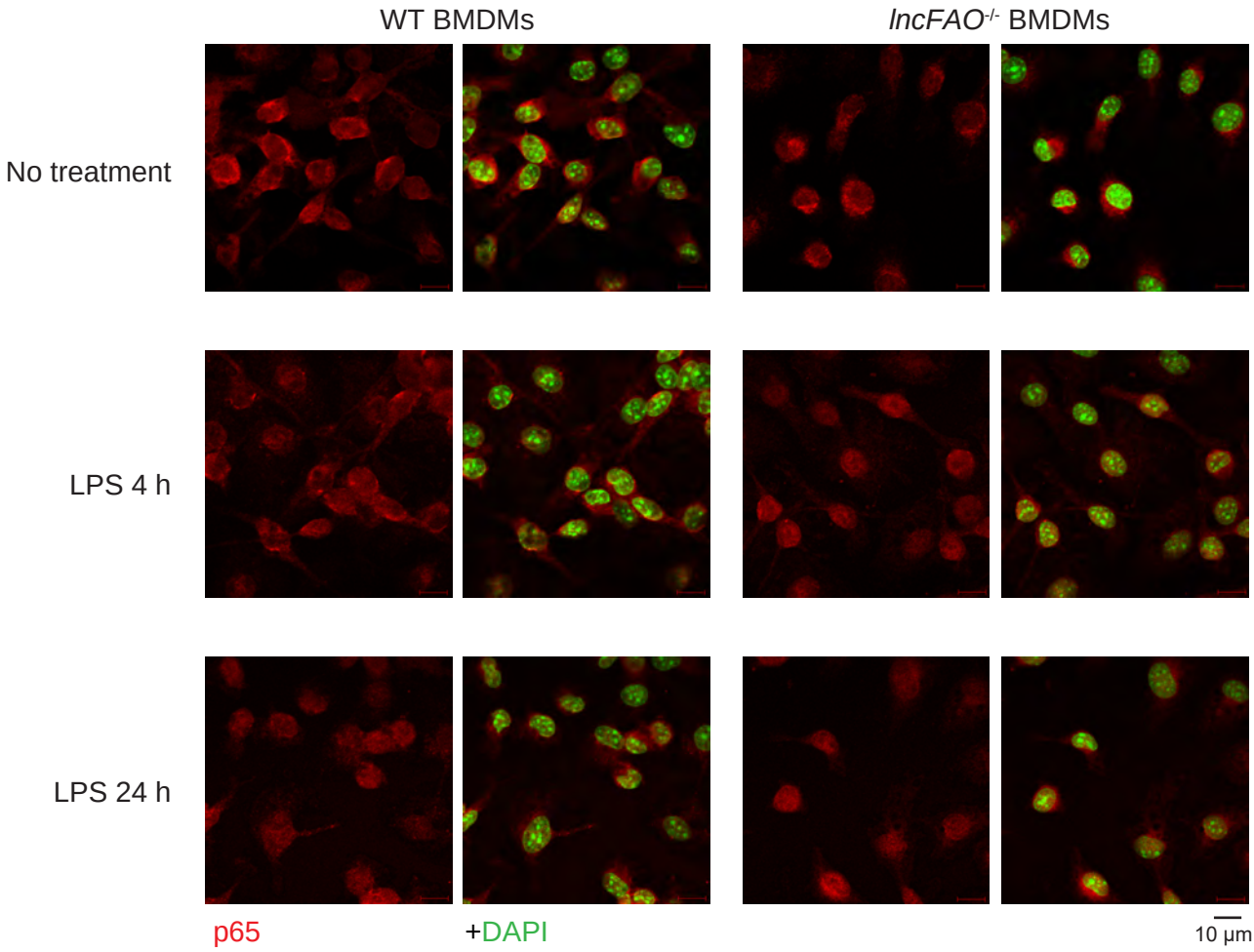






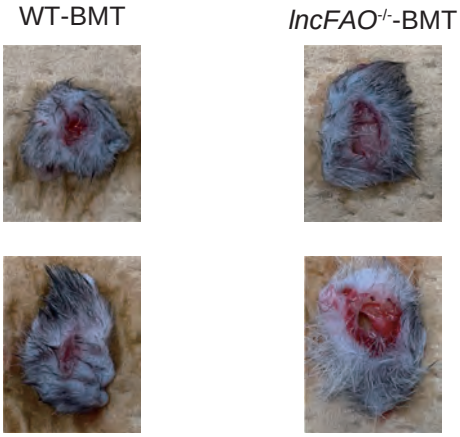


Supplementary Figure S4

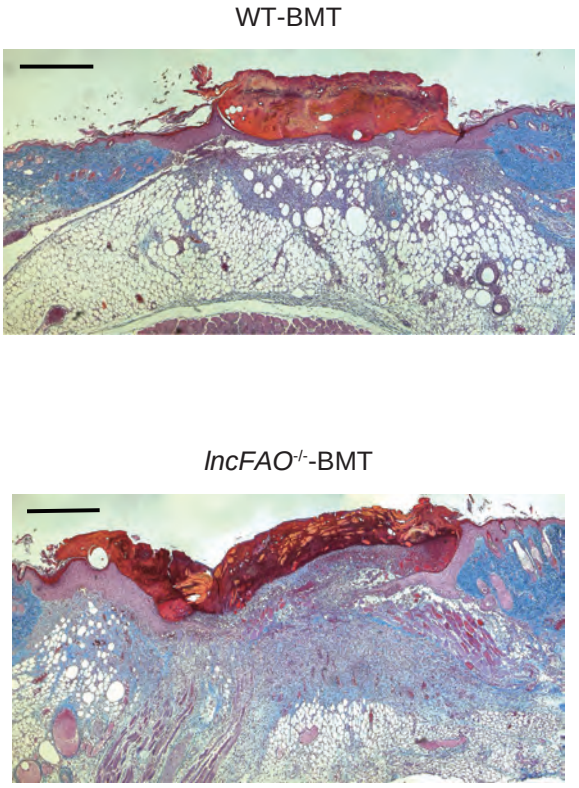


Supplementary Figure S5

A

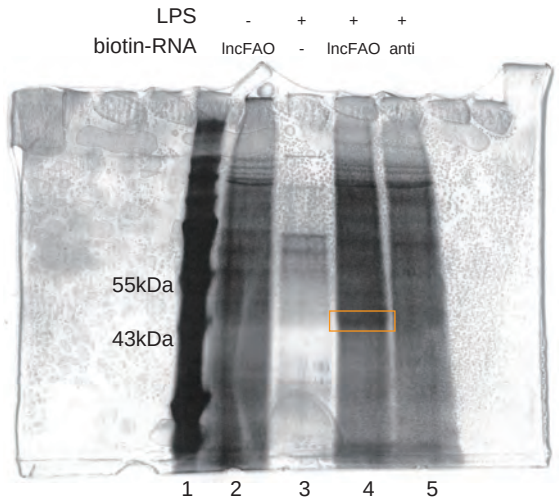


B



Supplementary Figure S6

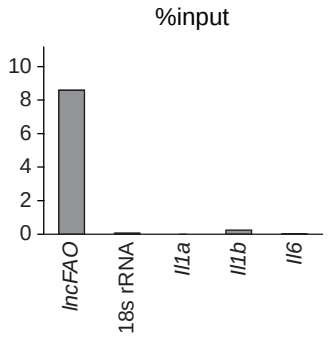
A



B

Protein	probability	Coverage	% spectra
HADHB	100%	36%	2.6%
hnRNP A/B	100%	11%	0.48%
Keratin	100%	4~29%	0.48~2.1%
Actin	100%	32~53%	2.7~4.2%

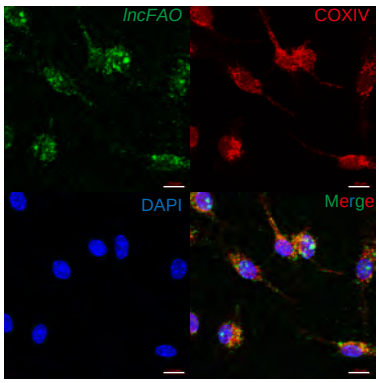
Supplementary Figure S7



Supplementary Figure S8

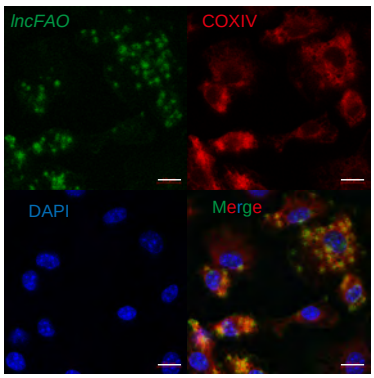
A

Untreated



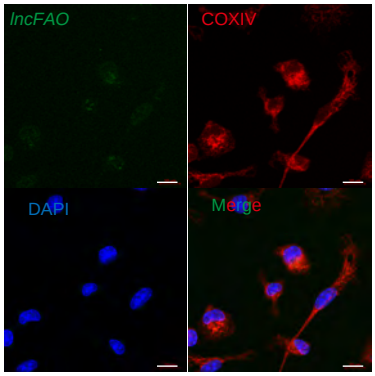
B

LPS 24 h



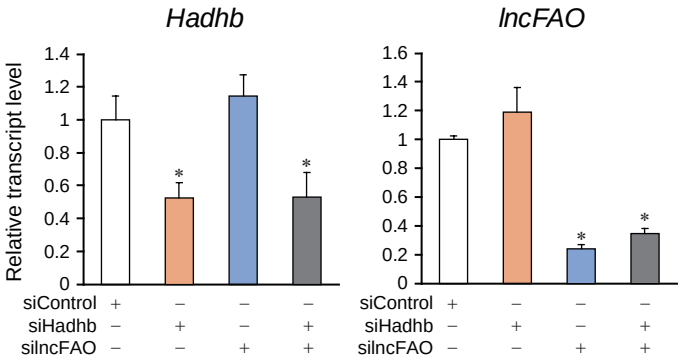
C

lncFAO$^{-/-}$ BMDM

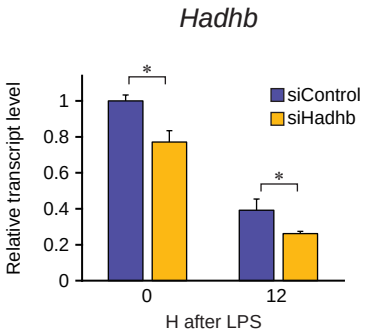


Supplementary Figure S9

A

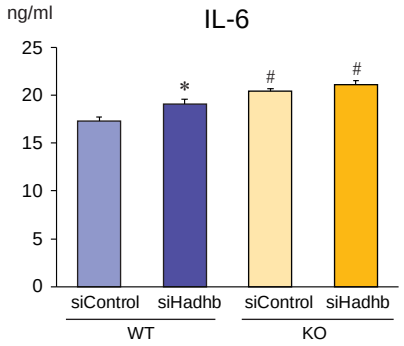


B

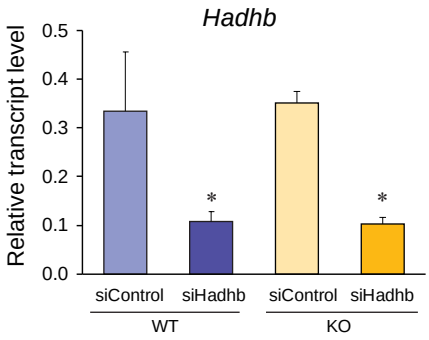


Supplementary Figures S10

A



B



Supplementary Figures S11