Supplementary Materials and Methods

Bacterial metagenomics and meta-transcriptomics analysis

Fecal bacteria genomic DNA was isolated from snap-frozen stool samples from the indicated strains using the PowerSoil DNA Isolation Kit (MoBio Laboratories, Carlsbad, CA, USA) following the manufacturer's protocol. The V4 region of the 16S rRNA gene was amplified with forward primer 515F containing the Illumina Flowcell adapter sequences and reverse primer 806R containing the Illumina Flowcell adapter sequences followed by barcode identifiers[1]. Gel-purified 16S amplifications were sequenced on an in-house Illumina MiSeq sequencing platform[1]. Sequence data was analyzed within the MacQIIME[2] and parsed based upon sample-specific barcodes and trimmed to a minimum quality score of 20. Operational taxonomic units (OTUs) at 97% were then picked against the Greengenes 13.5 database[3] using UCLUST[4] for taxonomic assignment. Beta diversity analyses were conducted by UNIFRAC[5].

Fecal total RNA was then isolated from snap-frozen stool samples using RNA PowerSoil® Total RNA Isolation Kit (MoBio Laboratories, Carlsbad, CA, USA) following the manufacturer's protocol. RNAseq libraries were constructed using the TruSeq Stranded RNA Sample Preparation Kit (Illumina, San Diego, CA, USA), and the pooled libraries were sequenced on an Illumina HiSeq2500 platform. The mouse sequence reads were removed and the rest of sequence reads were assigned to either a mouse meta-transcriptome database or the NCBI non-redundant protein database. The corresponding protein sequences were extracted from the meta-transcriptome reads using Megablast against the NCBI bacterial genome database, UBLAST against the Salvage: non-redundant protein database, and UBLAST against select mouse gut studies from MG-RAST, and then converted to clusters of orthologous groups (COGs) by

running against the COGs protein database. The iPath2 was used to group the COGs into the KEGG (Kyoto Encyclopedia of Genes and Genomes) pathways.

RNA sequencing analysis of the ileum of wild type and TLR4$^{\Delta IEC}$ mice

Total RNA was isolated from the ileum of TLR4$^{\Delta IEC}$ and wild type mice at the age of 24 weeks using the RNeasy mini kit following the manufacturer's protocol. The quality of the RNA samples was analyzed by Agilent Bioanalyzer 2100 (Agilent Technologies), and only RNA samples with an RNA integrity number greater than 7 were used for further analysis. For whole transcriptome library preparation, we first performed rRNA depletion using the GeneRead rRNA Depletion Kit (Qiagen, Valencia, CA, USA) according to the manufacturer's instructions. EERC (External RNA Controls Consortium) spike-in RNA controls were added to 500ng of the product before fragmentation with RNAse III. Fragmented RNA was then purified and the concentration assessed via fluorometric assay and size distribution verified on the Agilent 2100 Bioanalyzer Pico Chip. The Ion Total RNA-Seq kit v2 for Whole Transcriptomes (Life Technologies, Carlsbad, CA, USA) was used for RNA preparation, and strand-specific reverse transcription was performed with SuperScript® III Enzyme Mix followed by magnetic bead clean-up of the cDNA. The cDNA was amplified with a high fidelity polymerase with proof reading activity (Platinum® PCR SuperMix High Fidelity) for a total of 16 cycles, using primers that complete the adapter sequence and add barcode sequences to each sample, followed by a further round of magnetic bead purification. Yield and size distribution were assessed by fluorometric assay and Bioanalyzer High Sensitivity DNA Chip at each step. Barcoded libraries were then pooled and diluted for emulsion amplification on Ion Sphere™ Particles (ISPs) using the Ion One Touch 2 System (Life Technologies, Carlsbad, CA, USA). Enriched ISP particles were prepared for

sequencing by heat-denaturing the template cDNA and annealing the sequencing primer. QC reagents were removed from the chip and the ISPs were loaded, mixed and centrifuged for even ISP distribution into the wells of the high-density array.

Read files were mapped using Tophat v. 2.0.10[6,7] in conjunction with Bowtie2 v2.1.0[8]. The resulting .bam files were used for compiling Quality Control Statistics and analyzed using RNASeqnator v0.8 to determine Transcript-level and Exon-level average coverage values. To remove small RNA transcripts, transcript length distributions per sample were plotted, and transcripts with L < 223 were excluded. The coverage values were analyzed using caGEDA for data normalization[9] and G-Thresholding was used TO find differentially expressed genes. J5 values were calculated on normalized data for differentially expressed genes at transcript level [9]. Pathway analysis was then conducted on the differentially expressed genes using Ingenuity Pathway Analysis.

Quantification of food intake

The Comprehensive Laboratory Animal Monitoring System (CLAMS) (Columbus Instruments, Columbus, OH, USA) were used to determine the food intake. TLR4$^{\Delta IEC}$ and wild type mice were individually placed in CLAMS cages and food consumption was recorded every 30 minutes and the accumulated data over 24 hours in the last light/dark cycle were used as daily food intake.

Circulating hormone levels, cholesterol, triglycerides, and endotoxin

Twenty-four-week-old mice were fasted overnight and the blood was collected by periorbital bleeding puncture. After 30 minutes clotting at room temperature, the serum was collected at

2,000 x g for 10 minutes at 4 ^{o}C. The mouse leptin ELISA kit (Enzo Life Sciences, Farmingdale, NY, USA), mouse ghrelin ELISA kit (MyBioSource, San Diego, CA, USA), and mouse insulin ELISA kit (ALPCO, Salem, NH, USA) were used to quantify the circulating hormone levels, the Infinity™ cholesterol liquid stable reagent (Thermo Scientific, Halethorpe, MD, USA) and Infinity™ triglycerides liquid stable reagent (Thermo Scientific, Halethorpe, MD, USA) were used to quantify the total cholesterol and triglycerides, and the Pierce LAL chromogenic endotoxin quantitation kit (Thermo Scientific, Halethorpe, MD, USA) was used to quantify the endotoxin levels in the serum from TLR4$^{\Delta IEC}$ and wild type mice.

Measurement of hepatic triglycerides

Freshly harvested, snap-frozen liver samples were digested with ethanolic KOH overnight at 55°C. The digestion samples were mixed with 50% ethanol and the supernatants were mixed with $MgCl_2$. After 10-minute-incubation on ice, the saponified and neutralized supernatants were used to determine triglyceride concentrations using Infinity triglycerides measurement kit according to the manufacturer's instructions (Thermo Scientific, Halethorpe, MD, USA).

Determination of total bacterial content

The total DNA from mice colonic contents samples was isolated from snap-frozen stool samples using PowerSoil DNA Isolation Kit (MoBio Laboratories, Carlsbad, CA, USA) following the manufacturer's protocol, and 1 ng purified DNA was used as a template to determine the copy numbers of bacterial 16S rDNA on the Bio-Rad CFX96 Real-Time System (Biorad, Hercules, CA, USA) using the primers 338F (5'-ACTCCTACGGGAGGCAGCAG-3') and 518R (5'-ATTACCGCGGCTGCTGG-3') which amplify all the bacterial 16S rDNA[10]. The copy numbers

of bacterial 16S rDNA were normalized to the sample weight and the mean value of wild type mice without antibiotics treatment was set as 1.

Generation and confirmation of TLR4$^{\Delta Myeloid}$ mice.

The breeding scheme for the generation of TLR4$^{\Delta Myeloid}$ mice is shown in **Supplemental Figure 7A.** In brief, we first bred the LysM$^{cre/cre}$ mouse from Jackson Laboratory (B6.129P2-Lyz2$^{tm1(cre)Ifo}$/J) which expresses the full length *Tlr4* and which we now denote as TLR4$^{wt/wt}$;LysM$^{cre/cre}$ with our recently generated TLR4$^{loxP/loxP}$ mouse[11] expressing full length LysM, which we denote as TLR4$^{loxP/loxP}$;LysM$^{wt/wt}$ mice. This breeding generates male stud mice ie TLR4$^{loxP/wt}$; LysM$^{cre/wt}$ mice. These stud mice were then back-bred with the TLR4$^{loxP/loxP}$;LysM$^{wt/wt}$ to generate 4 progeny, namely wild type mice (i.e. TLR4$^{loxP/loxP}$; LysM$^{wt/wt}$) , TLR4$^{\Delta Myeloid}$ mice (i.e. TLR4$^{loxP/loxP}$; LysM$^{cre/WT}$) additional stud mice (i.e. TLR4$^{loxP/wt}$; LysM$^{cre/WT}$) and TLR4$^{loxP/wt}$; LysM$^{wt/wt}$ mice in a ratio of 25%: 25%: 25%: 25%. Further experiments were performed on the TLR4$^{\Delta Myeloid}$ mice. Confirmation that the peritoneal macrophages in the TLR4$^{-/-}$ and TLR4$^{\Delta Myeloid}$ mice lack TLR4 is shown in **Supplemental Figure 7B**. For these studies, peritoneal macrophages were isolated from wild type, TLR4$^{-/-}$ and TLR4$^{\Delta Myeloid}$ mice by lavage in 5 ml of ice-cold saline followed by gentle centrifugation, and the mRNA expression of *Tlr4* and the housekeeping gene *Rplp0* were determined using mouse *Tlr4*-specific primers (forward: 5'-CAGCAAAGTCCCTGATGACA-3'; reverse: 5'-TCCAGCCACTGAAGTTCTGA-3') and *Rplp0*-specfic primers (forward: 5'-GGCGACCTGGAAGTCCAACT-3'; reverse: 5'-CCATCAGCACCACAGCCTTC-3'), respectively. To further validate that the myeloid cells do not express functional TLR4 protein, the peritoneal macrophages isolated from wild type and TLR4$^{\Delta Myeloid}$ mice were treated with

TLR4 ligand LPS (100 ng/mL for 2 hours), and the expression of TLR4 signaling downstream genes *IL-6* and *TNF-alpha,* as well as the expression of TLR4 and LysM, were quantified using qPCR (**Supplemental Figure 7B-C)**.

Supplemental Figure Legends

Supplemental Figure 1. ***Determination of food intake, serum appetite hormones, hepatic triglycerides, adipocyte size, serum insulin, cholesterol, triglycerides and endotoxin in wild type and TLR4$^{\Delta IEC}$ mice.*** **A**: Food intake of wild type (n=5) and TLR4$^{\Delta IEC}$ (n=5) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **B**: Concentration of serum leptin (i) and ghrelin (ii) of wild type (n=12 and 9, respectively) and TLR4$^{\Delta IEC}$ (n=12 and 7, respectively) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **C**: Hepatic triglycerides content of wild type (n=5) and TLR4$^{\Delta IEC}$ (n=5) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **D**: Adipocyte size of wild type (n=6) and TLR4$^{\Delta IEC}$ (n=7) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **E**: Serum insulin levels of wild type (n=9) and TLR4$^{\Delta IEC}$ (n=8) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **F**: Serum total cholesterol levels of wild type (n=11) and TLR4$^{\Delta IEC}$ (n=9) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **G**: Serum total triglycerides levels of wild type (n=11) and TLR4$^{\Delta IEC}$ (n=7) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse. **H**: Serum endotoxin levels of wild type (n=5) and TLR4$^{\Delta IEC}$ (n=5) mice fed standard chow at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse.

Supplemental Figure 2. ***The administration of a high fat diet results in the development of obesity and metabolic syndrome in mice lacking TLR4 on the intestinal epithelium.*** **A**: **i**: Determination of body weight between 3 and 12 weeks of wild type (n=13) and TLR4$^{\Delta IEC}$ (n=21) mice who were fed high fat diet (HFD); **ii**: body weight at 12 weeks of wild type (n=13) and TLR4$^{\Delta IEC}$ (n=21) mice. Data are represented as mean ± SEM; *p<0.05 wild type vs TLR4$^{\Delta IEC}$ mice at the indicated time point; each symbol represents a separate mouse. **B**: The glucose tolerance test of wild type (n=13) and TLR4$^{\Delta IEC}$ (n=21) mice fed high fat diet at the age of 12 weeks is shown. Data are represented as mean ± SEM; *p<0.05 wild type vs. TLR4$^{\Delta IEC}$ mice. **C**: Weight of inguinal fat, mesenteric fat and liver of wild type (n=7 to 9) and TLR4$^{\Delta IEC}$ (n=6) mice fed high fat diet at the age of 12 weeks. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse. **D**: Serum total cholesterol levels of wild type (n=6) and TLR4$^{\Delta IEC}$ (n=6) mice fed high fat diet at the age of 24 weeks. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse. **E**: Serum total triglycerides levels of wild type (n=6) and TLR4$^{\Delta IEC}$ (n=6) mice fed high fat diet at the age of 24 weeks. Data are represented as mean ± SEM; each symbol represents a separate mouse.

Supplemental Figure 3. ***Oral glucose tolerance test of wild-type and TLR4$^{\Delta IEC}$ mice at the age of 12 weeks.*** The glucose tolerance test of wild type (n=4) and TLR4$^{\Delta IEC}$ (n=5) mice fed standard chow at the age of 12 weeks is shown. Data are represented as mean ± SEM**.**

Supplemental Figure 4. ***Quantification of bacterial load and differentially expressed genes in the PPAR signaling pathway.*** **A**: qPCR showing the bacterial content in the ileum of wild type

(n=7 and 8, respectively) and TLR4$^{\Delta IEC}$ (n=6 and 6, respectively) mice treated without and with antibiotics. All data were normalized to the weight of colonic contents, and the relative 16S copy number in wild type mice was set to 1. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse. **B**: Body weight at 24 weeks of wild type (n=10) and TLR4$^{\Delta IEC}$ (n=8) mice fed standard chow and co-housed. Data are represented as mean ± SEM; each symbol represents a separate mouse. **C**: Weight of inguinal fat and liver at the age of 24 weeks of wild type (n=10) and TLR4$^{\Delta IEC}$ (n=8) mice fed standard chow and co-housed. Data are represented as mean ± SEM; each symbol represents a separate mouse. **D:** The glucose tolerance test at the age of 24 weeks of wild type (n=8) and TLR4$^{\Delta IEC}$ (n=8) mice fed standard chow and co-housed is shown. Data are represented as mean ± SEM; *p<0.05 wild type vs. TLR4$^{\Delta IEC}$ mice.

Supplemental Figure 5. ***Quantification of antimicrobial peptide lysozyme in the ileum of wild-type and TLR4$^{\Delta IEC}$ mice.*** The mRNA expression of *lysozyme* in the ileum of wild type (n=5 and 7, respectively) and TLR4$^{\Delta IEC}$ (n=6 and 6, respectively) mice fed standard chow or administrated with antibiotics. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse.

Supplemental Figure 6. ***Mild inflammation and altered expression of PPAR-regulated genes in the ileum of wild-type and TLR4$^{\Delta IEC}$ mice.*** **A**: RNA sequencing identifying differentially expressed PPAR genes between wild type and TLR4$^{\Delta IEC}$ mice. The red color shows genes with increased expression and blue color shows those with decreased expression in the ileum of

TLR4$^{\Delta IEC}$ mice compared with wild type mice. **B**: The mRNA expression of *Ppara* and *Pparg* in the ileum of wild type (n=9 and 6, respectively) and TLR4$^{\Delta IEC}$ (n=8 and 8, respectively) mice. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; each symbol represents a separate mouse. **C**: qPCR showing the expression of PPAR family genes *Fiaf*, *Fatp1*, *Hmgcs2* and *Fabp1* in the ileum of wild type (n=8) and TLR4$^{\Delta Myeloid}$ (n=4) mice fed standard chow at the age of 24 weeks. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; each symbol represents a separate mouse. **D**: The mRNA expression of macrophage markers *F4/80*, *Cd68* and *Mcp1* in the ileum of wild type (n=6) and TLR4$^{\Delta IEC}$ (n=10) mice fed standard chow. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse. **E**: The mRNA expression of neutrophil markers *Mpo* and *Elane* in the ileum of wild type (n=7) and TLR4$^{\Delta IEC}$ (n=8) mice fed standard chow. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; *p<0.05 for the indicated comparison; each symbol represents a separate mouse. **F**: The mRNA expression of *Il-6* in the ileum of wild type (n=7) and TLR4$^{\Delta IEC}$ (n=9) mice fed standard chow. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; each symbol represents a separate mouse.

Supplemental Figure 7. ***Generation and confirmation of*** TLR4$^{\Delta Myeloid}$ mice. A: Breeding scheme for the generation of TLR4$^{\Delta Myeloid}$ mice. In brief, we first bred the LysM$^{cre/cre}$ mouse from

Jackson Laboratory (B6.129P2-Lyz2$^{tm1(cre)Ifo}$/J) which expresses the full length *Tlr4* and which we now denote as TLR4$^{wt/wt}$;LysM$^{cre/cre}$ with our recently generated TLR4$^{loxP/loxP}$ mouse[11] expressing full length LysM, which we denote as TLR4$^{loxP/loxP}$;LysM$^{wt/wt}$ mice. This breeding generates male stud mice ie TLR4$^{loxP/wt}$; LysM$^{cre/wt}$ mice. These stud mice were then back-bred with the TLR4$^{loxP/loxP}$;LysM$^{wt/wt}$ to generate 4 progeny, namely wild type mice (i.e. TLR4$^{loxP/loxP}$; LysM$^{wt/WT}$), TLR4$^{\Delta Myeloid}$ mice (i.e TLR4$^{loxP/loxP}$; LysM$^{cre/wt}$) additional stud mice (i.e. TLR4$^{loxP/wt}$; LysM$^{cre/wt}$) and TLR4$^{loxP/wt}$; LysM$^{wt/wt}$ mice in a ratio of 25%: 25%: 25%: 25% B: Verification of TLR4$^{\Delta Myeloid}$ mice mice genotype in peritoneal macrophages. (1) Reverse transcription PCR detection of *Tlr4* and *Rplp0* mRNA (**in Bi**) and lysM (**in Bii**) from the peritoneal macrophages of wild type (n=2), TLR4$^{\Delta Myeloid}$ mice (n=2) and TLR4$^{\Delta Myeloid}$ mice (n=2). (ii) qPCR quantification of LysM gene expression from the peritoneal macrophages of wild type (n=6), TLR4$^{\Delta Myeloid}$ mice (n=3), stud mice (n=4) and LysM^{cre} mice (n=6). All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in wild type mice was set to 1. Data are represented as mean ± SEM; *$p<0.05$ for the indicated comparison; each symbol represents a separate mouse. **C:** The peritoneal macrophages of wild type (n=2) and TLR4$^{\Delta Myeloid}$ (n=3) mice were treated with TLR4 agonist lipopolysaccharide (100 ng/ml for 6 h), and the mRNA expression of *Il-6* (i) and *Tnf* (ii) was shown. All data were normalized to the mRNA expression of *Rplp0*, and the mRNA expression in saline-treated samples was set to 1. Data are represented as mean ± SEM; *$p<0.05$ for the indicated comparison; each symbol represents a separate mouse.

Supplementary references

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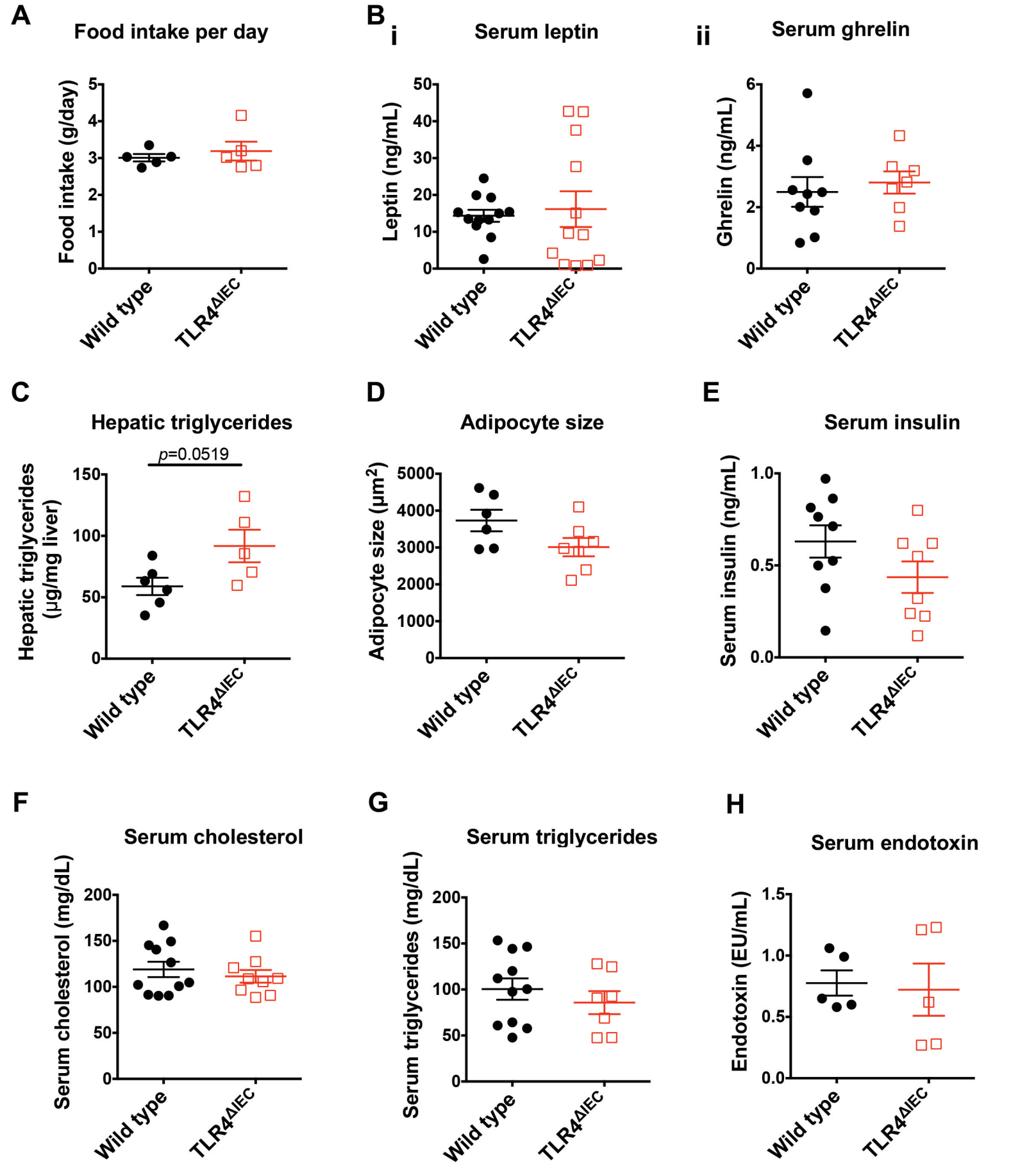
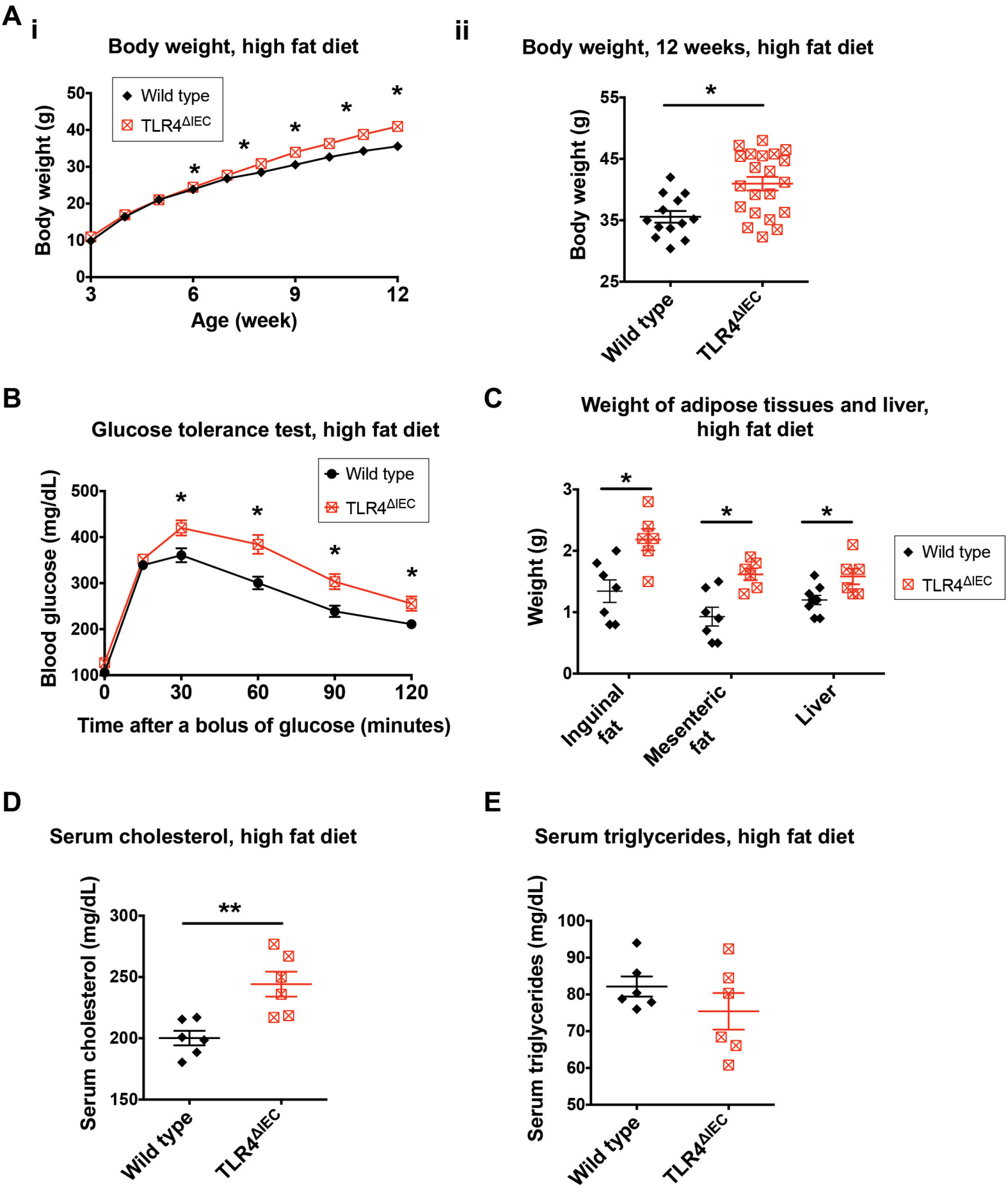
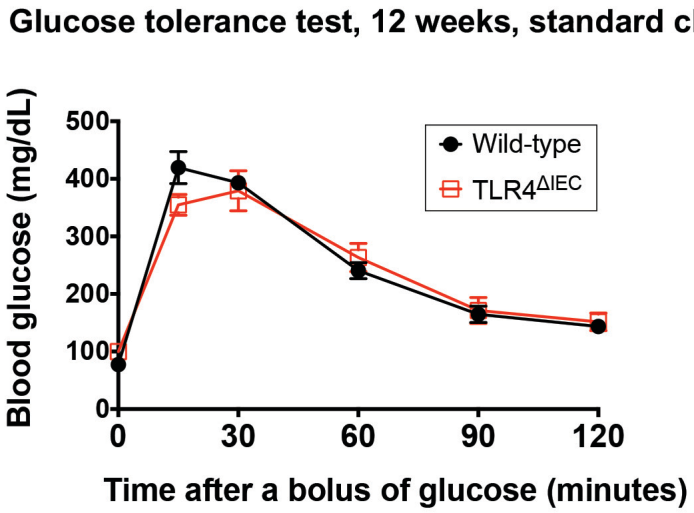

A
Food intake per day
Food intake (g/day)
0
1
2
3
4
5
Wild type
TLR4ΔIEC
B
i
Serum leptin
Leptin (ng/mL)
0
10
20
30
40
50
Wild type
TLR4ΔIEC
ii
Serum ghrelin
Ghrelin (ng/mL)
0
2
4
6
Wild type
TLR4ΔIEC
C
Hepatic triglycerides
p=0.0519
Hepatic triglycerides (µg/mg liver)
0
50
100
150
Wild type
TLR4ΔIEC
D
Adipocyte size
Adipocyte size (µm2)
0
1000
2000
3000
4000
5000
Wild type
TLR4ΔIEC
E
Serum insulin
Serum insulin (ng/mL)
0.0
0.5
1.0
Wild type
TLR4ΔIEC
F
Serum cholesterol
Serum cholesterol (mg/dL)
0
50
100
150
200
Wild type
TLR4ΔIEC
G
Serum triglycerides
Serum triglycerides (mg/dL)
0
50
100
150
200
Wild type
TLR4ΔIEC
H
Serum endotoxin
Endotoxin (EU/mL)
0.0
0.5
1.0
1.5
Wild type
TLR4ΔIEC


A
i
Body weight, high fat diet
Wild type
TLR4ΔIEC
Body weight (g)
Age (week)
ii
Body weight, 12 weeks, high fat diet
Body weight (g)
Wild type
TLR4ΔIEC
B
Glucose tolerance test, high fat diet
Wild type
TLR4ΔIEC
Blood glucose (mg/dL)
Time after a bolus of glucose (minutes)
C
Weight of adipose tissues and liver, high fat diet
Weight (g)
Inguinal fat
Mesenteric fat
Liver
Wild type
TLR4ΔIEC
D
Serum cholesterol, high fat diet
Serum cholesterol (mg/dL)
Wild type
TLR4ΔIEC
E
Serum triglycerides, high fat diet
Serum triglycerides (mg/dL)
Wild type
TLR4ΔIEC


Glucose tolerance test, 12 weeks, standard chow
Blood glucose (mg/dL)
500
400
300
200
100
0
0
30
60
90
120
Time after a bolus of glucose (minutes)
Wild-type
TLR4ΔIEC

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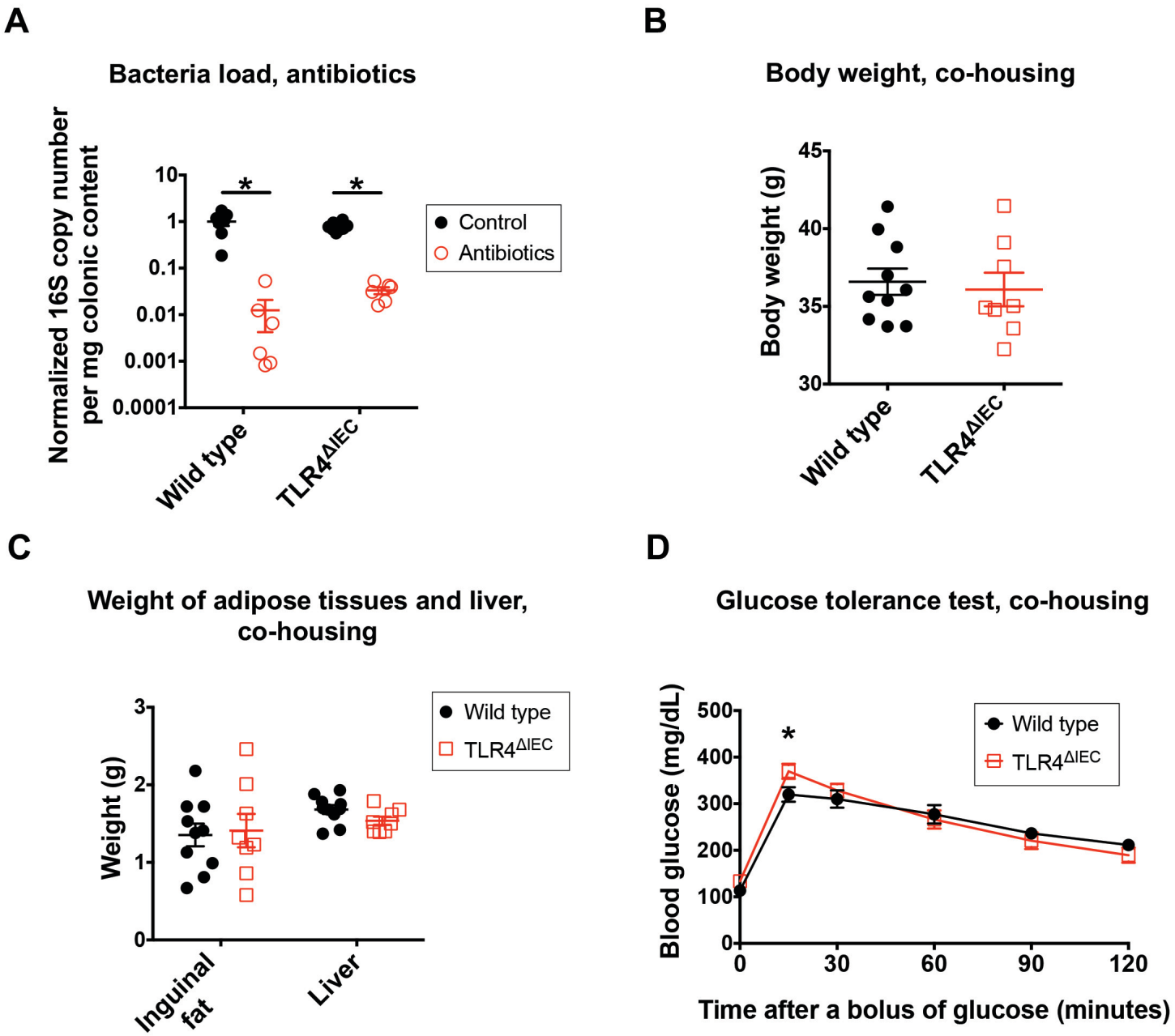

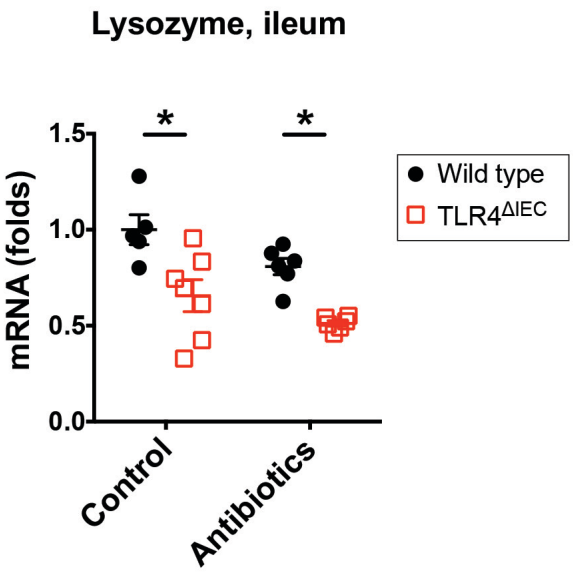
Lysozyme, ileum
mRNA (folds)
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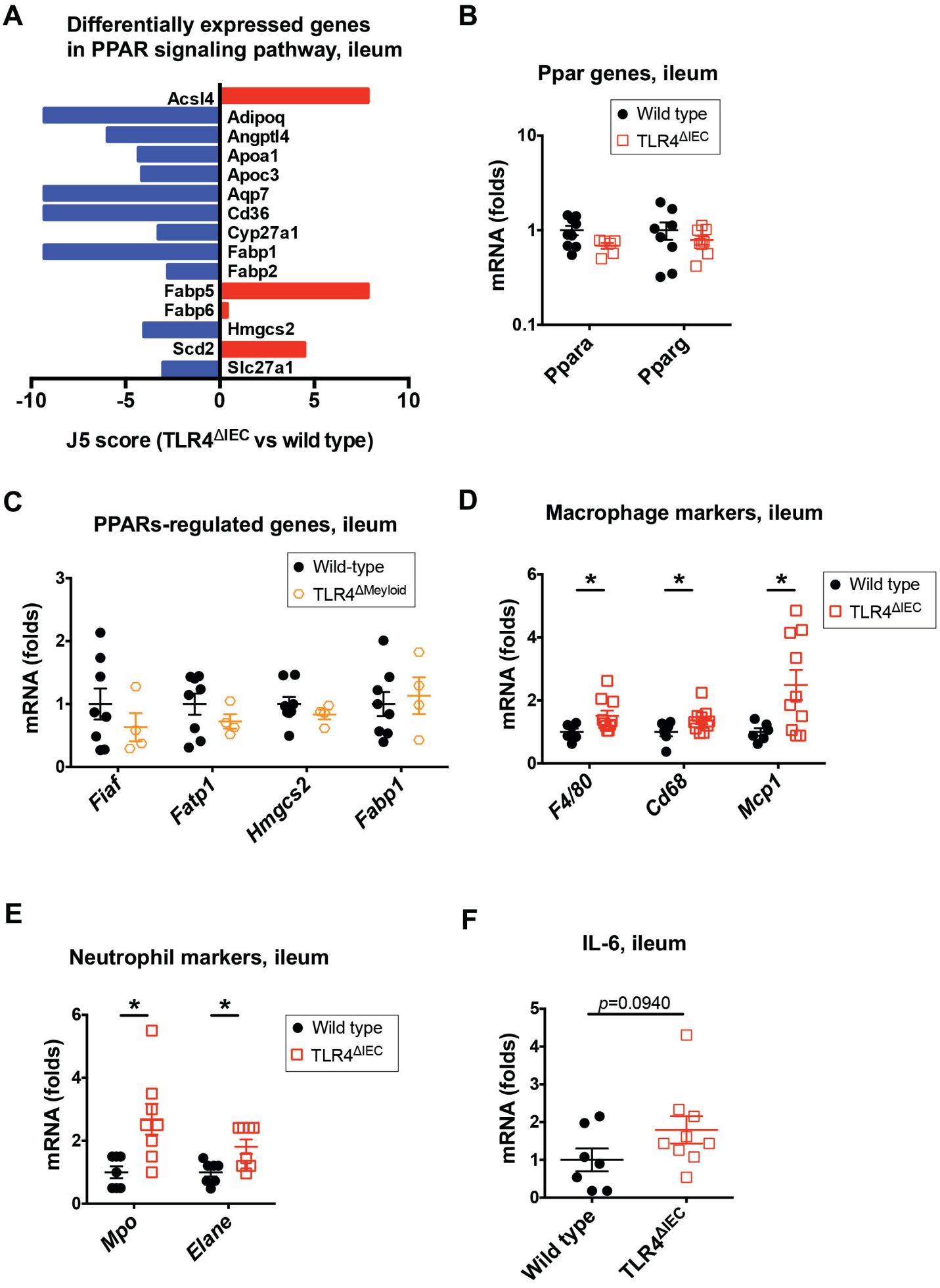
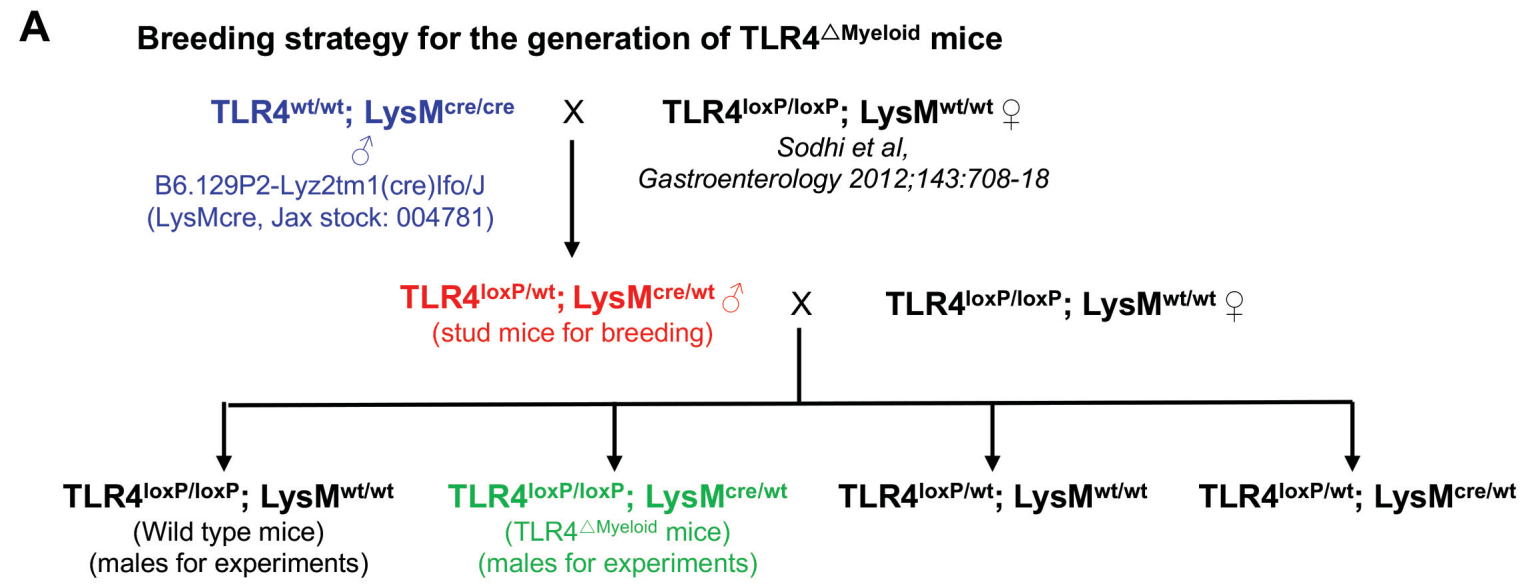
0.5
0.0
*
*
Wild type
TLR4ΔIEC
Control
Antibiotics

Supplemental Figure 6 Lu et al.

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Breeding strategy for the generation of TLR4△Myeloid mice
TLR4wt/wt; LysMcre/cre ♂
B6.129P2-Lyz2tm1(cre)Ifo/J
(LysMcre, Jax stock: 004781)
X
TLR4loxP/loxP; LysMwt/wt ♀
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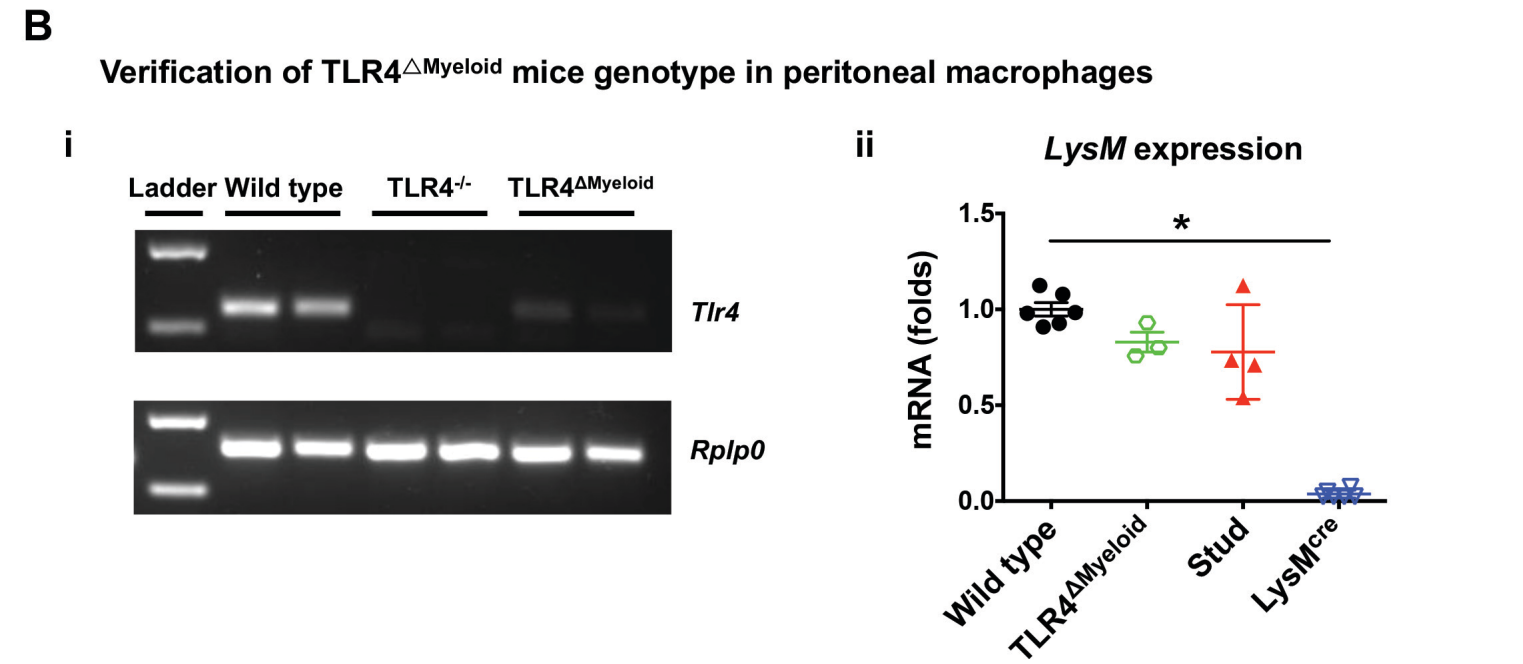
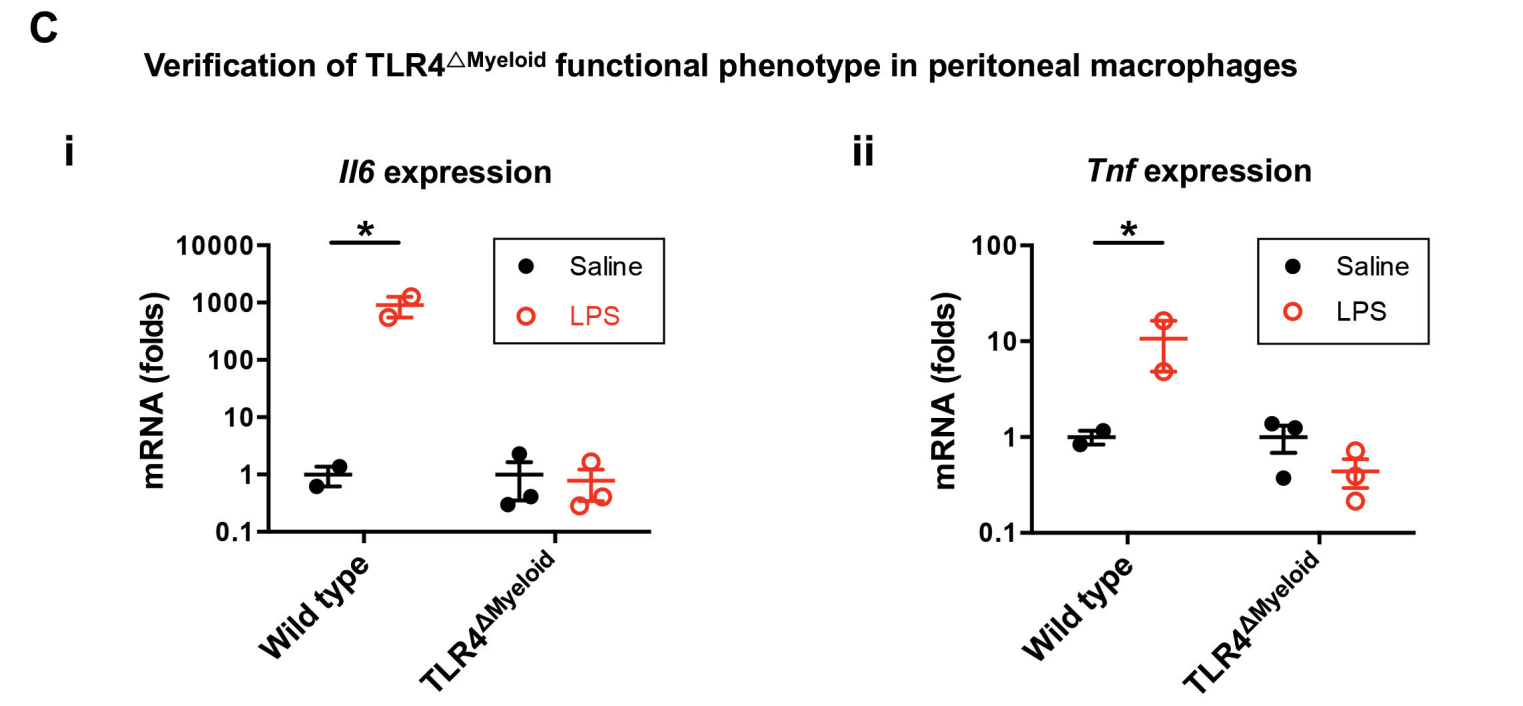
Gastroenterology 2012;143:708-18
TLR4loxP/wt; LysMcre/wt ♂
(stud mice for breeding)
X
TLR4loxP/loxP; LysMwt/wt ♀
TLR4loxP/loxP; LysMwt/wt
(Wild type mice)
(males for experiments)
TLR4loxP/loxP; LysMcre/wt
(TLR4△Myeloid mice)
(males for experiments)
TLR4loxP/wt; LysMwt/wt
TLR4loxP/wt; LysMcre/wt


B
Verification of TLR4△Myeloid mice genotype in peritoneal macrophages
i
Ladder
Wild type
TLR4-/-
TLR4ΔMyeloid
Tlr4
Rplp0
ii
LysM expression
mRNA (folds)
1.5
1.0
0.5
0.0
*
Wild type
TLR4ΔMyeloid
Stud
LysMcre


C
Verification of TLR4△Myeloid functional phenotype in peritoneal macrophages
i
Il6 expression
mRNA (folds)
10000
1000
100
10
1
0.1
*
Saline
LPS
Wild type
TLR4ΔMyeloid
ii
Tnf expression
mRNA (folds)
100
10
1
0.1
*
Saline
LPS
Wild type
TLR4ΔMyeloid