

Additional File 1

Figure Legends

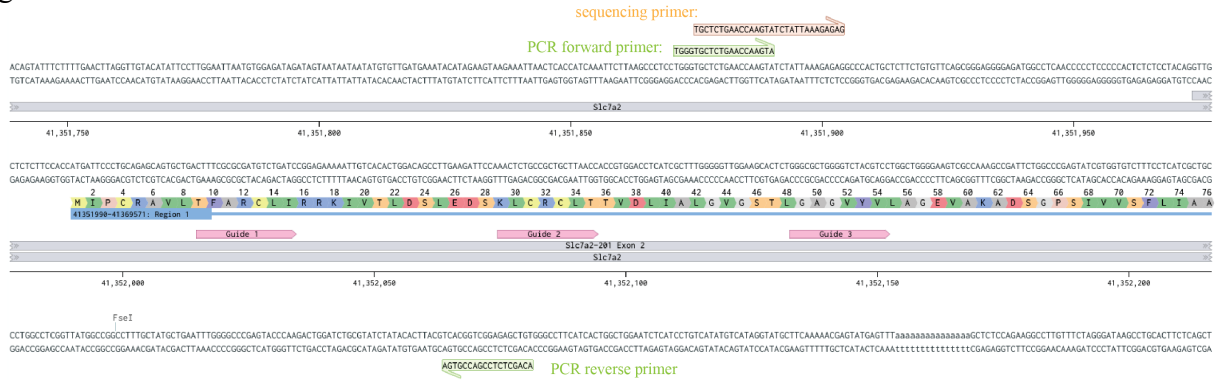
Fig. S1. Knocking out SLC7A2 by the CRISPR-Cas9 system. **A.** Scheme showing the three sgRNA sequence targeting exon 2 of the mouse *Slc7a2* gene (pink, guide 1, 2 and 3), the PCR primers for amplification (green, forward and reverse primers) and the sequencing primers for Sanger sequencing (orange, sequencing primer). **B.** Confirmation of the large fragment deletion in the SLC7A2KO clones in STHdhQ7 and Q111 cells with Sanger sequencing followed by ICE analysis using the online tool from Synthego. **C.** Intracellular NO levels monitored in real-time using DAF-2DA in STHdhQ7, Q7-SLC7A2KOs, Q111 and Q111-SLC7A2KO in the presence or absence of IFN γ /LPS treatment.

Fig. S2. RNA-seq analysis of transcriptomic changes in STHdhQ7 and Q111 cells in response to IFN γ /LPS treatment. **A.** PCA plot of all the samples used in the RNA-seq analysis. Q7-C: STHdhQ7 untreated; Q7-C: STHdhQ7 cells with 24-hr IFN γ /LPS treatment; Q111-C: STHdhQ111 untreated; Q111-C: STHdhQ111 cells with 24-hr IFN γ /LPS treatment. **B-C.** KEGG pathway enrichment analysis in STHdhQ7 (**B**) and Q111 (**C**) cells in response to IFN γ /LPS treatment.

Fig. S3. Mitochondrial fragmentation in STHdhQ111 cells in response to IFN γ /LPS treatment. STHdhQ7 and Q111 cells were transfected with mito-turboRFP and treated with IFN γ /LPS for 24 hours in the presence or absence of 1400W hydrochloride. Cells were then fixed in 4% paraformaldehyde with 4% sucrose to preserve mitochondrial morphology. Images were taken with a 60X oil objective (CFI Plan Apochromat Lambda 60X Oil, numerical aperture =1.4) using a laser scanning confocal microscope (Nikon A1R). Scale bar: 5 μ m.

Fig. S1.

A.



B.



C.

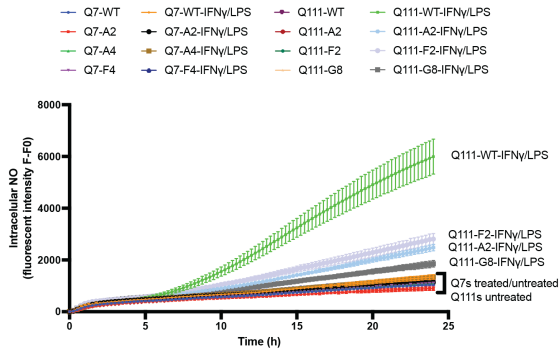
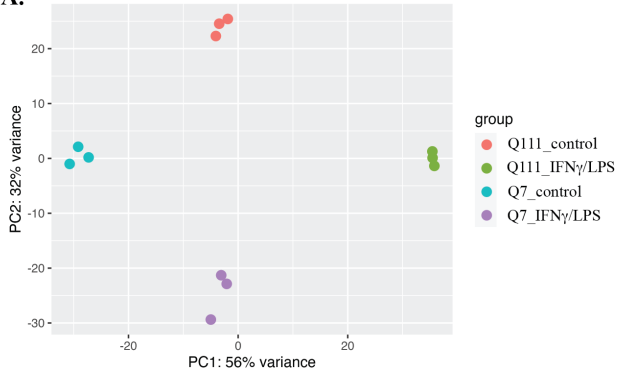
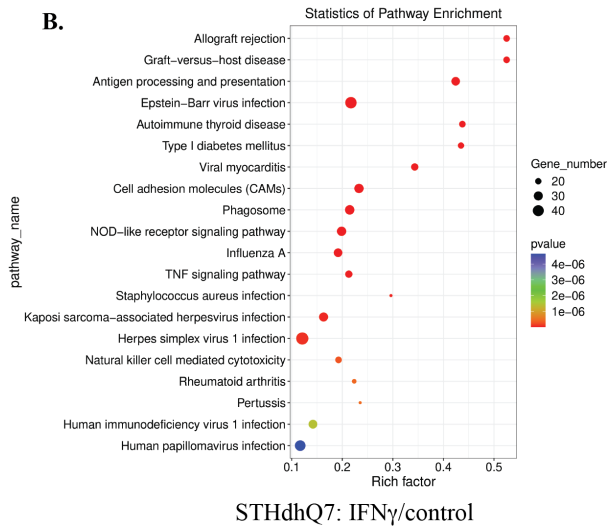


Fig. S2.

A.



B.



C.

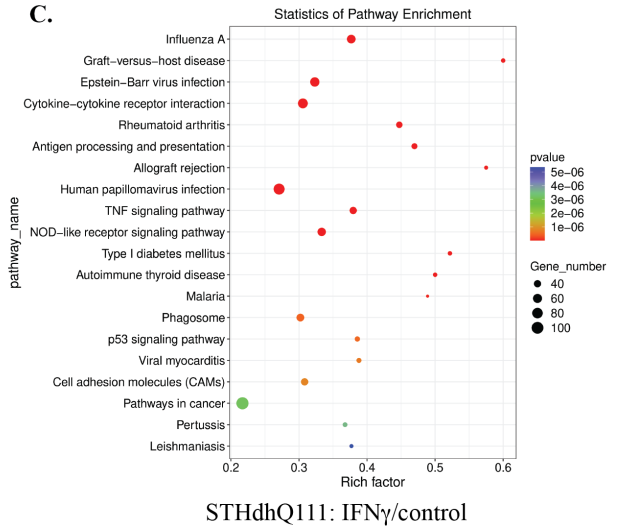


Fig. S3.

