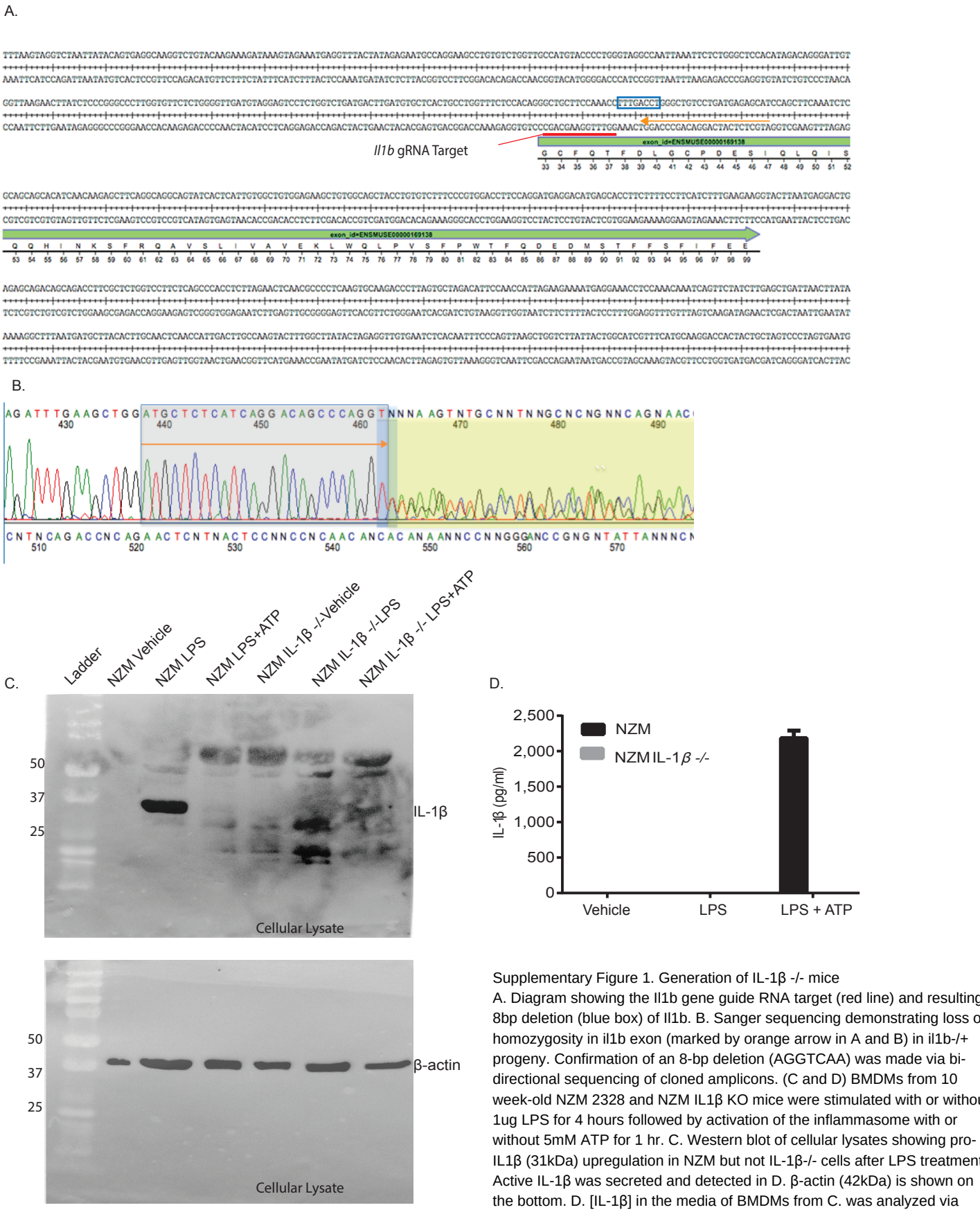
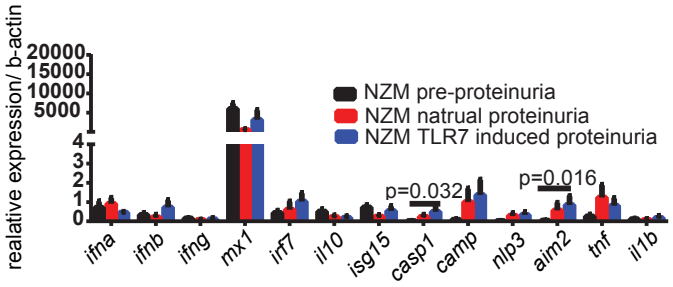


Supplementary Figure 1. Generation of IL-1β -/- mice
A. Diagram showing the Il1b gene guide RNA target (red line) and resulting 8bp deletion (blue box) of Il1b. B. Sanger sequencing demonstrating loss of homozygosity in il1b exon (marked by orange arrow in A and B) in il1b-/+ progeny. Confirmation of an 8-bp deletion (AGGTCAA) was made via bi-directional sequencing of cloned amplicons. (C and D) BMDMs from 10 week-old NZM 2328 and NZM IL1β KO mice were stimulated with or without 1ug LPS for 4 hours followed by activation of the inflammasome with or without 5mM ATP for 1 hr. C. Western blot of cellular lysates showing pro-IL1β (31kDa) upregulation in NZM but not IL-1β-/- cells after LPS treatment. Active IL-1β was secreted and detected in D. β-actin (42kDa) is shown on the bottom. D. [IL-1β] in the media of BMDMs from C. was analyzed via ELISA. No secreted IL-1β was detected from NZMIL-1β-/- mice.



Supplementary Figure 2. Changes in inflammatory response genes in TLR7-induced nephritis vs. natural nephritis
RNA was isolated from the kidney of 10 weeks-old NZM2328 mice stimulated with R848 until proteinuria(4+ by dipstick) development, from NZM2328 mice that naturally developed proteinuria (4+ by dipstick) at around 35 weeks of age, and NZM2328 mice that were pre-proteinuric (trace by dipstick). Real- time PCR was completed for analysis of the genes listed. Graphs display the mean+ SD for each gene as compared to the average of β-actin. n=4 for R848 treated, n=5 for aged NZM mice and n=5 for NZM pre-proteinurc mice. $p<0.05$ is considered significant via unpaired T test.