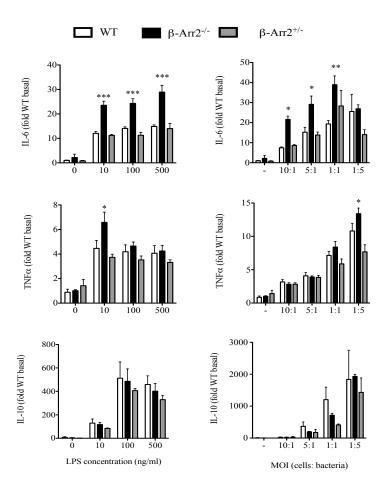


Supplementary figure 1: Representatives blot for MAPK and NFkB kinase activation in the lung following polymicrobial infection: Representative blots for data shown in Fig 4 for (A) naïve mice of three genotypes versus infected WT mice. (B) Infected mice of all three genotypes.

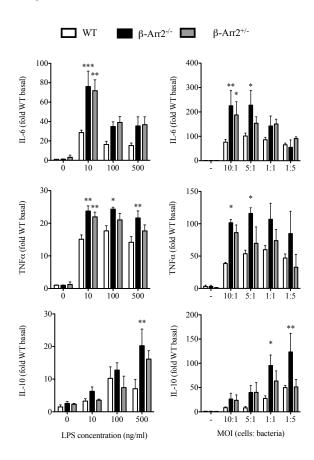
Figure S2



Supplementary figure 2: β-Arrestin2 negatively regulates cytokine production in resident peritoneal cell population: Resident peritoneal from naïve wild type (WT), β-arrestin-2 homozygous knockout (β-arr2^{-/-}) and β-arrestin-2 heterozygous (β-arr2^{+/-}) mice were obtained, processed and plated as described in the methods. The composition was similar in all three genotypes as determined by flow cytometric analysis. Cells were then stimulated with LPS and polymicrobial culture at different concentrations and multiplicity of infection (MOI) respectively. Cells were stimulated for 18 hours and supernatants assayed for IL-6, IL-10 and TNFα concentrations. Cytokine levels were

transformed as fold over WT basal. *p<0.05; **p<0.01; ***p<0.001 compared to WT as determined by 2-way ANOVA followed by Bonferroni post test. N=4-5 mice for each genotype.

Figure S3



Supplementary figure 3: β-Arrestin2 negatively regulates cytokine production in **splenocytes:** Splenocytes from naïve wild type (WT), β-arrestin-2 homozygous knockout (β-arr2^{-/-}) and β-arrestin-2 heterozygous (β-arr2^{+/-}) mice were processed and plated as described in the methods. The composition was similar in all three genotypes as determined by flow cytometric analysis. Cells were then stimulated with LPS and polymicrobial culture at different concentrations and multiplicity of infection (MOI) respectively. Cells were stimulated for 18 hours and supernatants assayed for IL-6, IL-10

and TNF α concentrations. Cytokine levels were transformed as fold over WT basal. *p<0.05; **p<0.01; ***p<0.001 compared to WT as determined by 2 -way ANOVA followed by Bonferroni post test. N=4-5 mice for each genotype