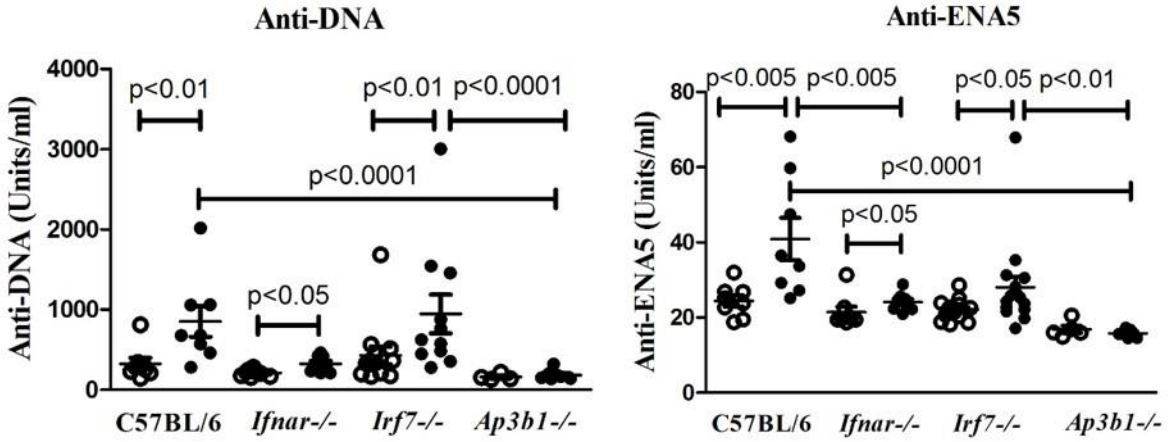
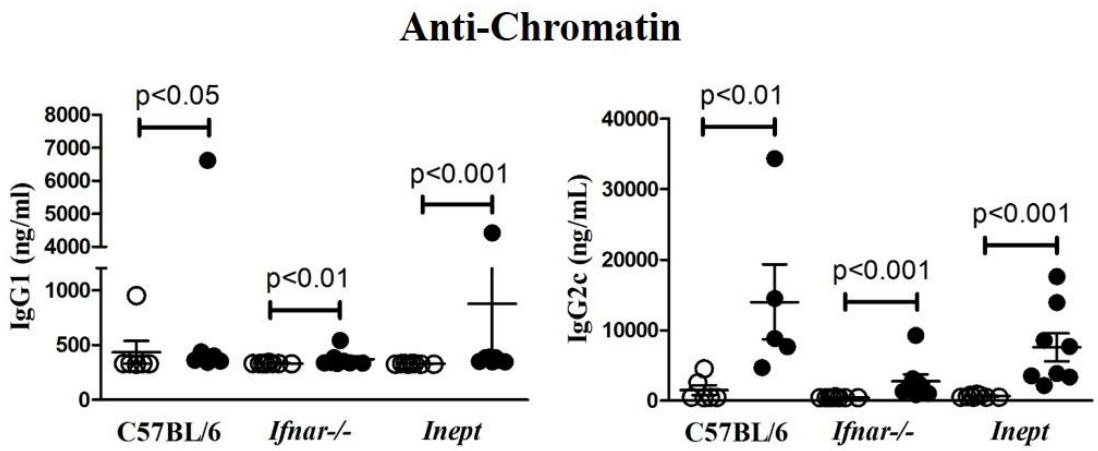
Supplemental Figure 1



Supplemental Figure 1. Anti-DNA and -ENA5 autoantibodies in mutant and wild type B6 mice after exposure to $HgCl_2$.

Dot plots of serum anti-DNA and -ENA5 antibodies in mice deficient for type I IFN receptos (*Ifnar1-/-*), IRF-7 (Irf7-/-), or AP3B1 (*Ap3b1-/-*) after a 4-week exposure to 40 μg $HgCl_2$ s.c. twice-a-week. The corresponding B6 (C57BL/6) wild type control was included for each assay. Anti-DNA and -ENA5 levels were measured by ELISA. P<0.05 are shown.

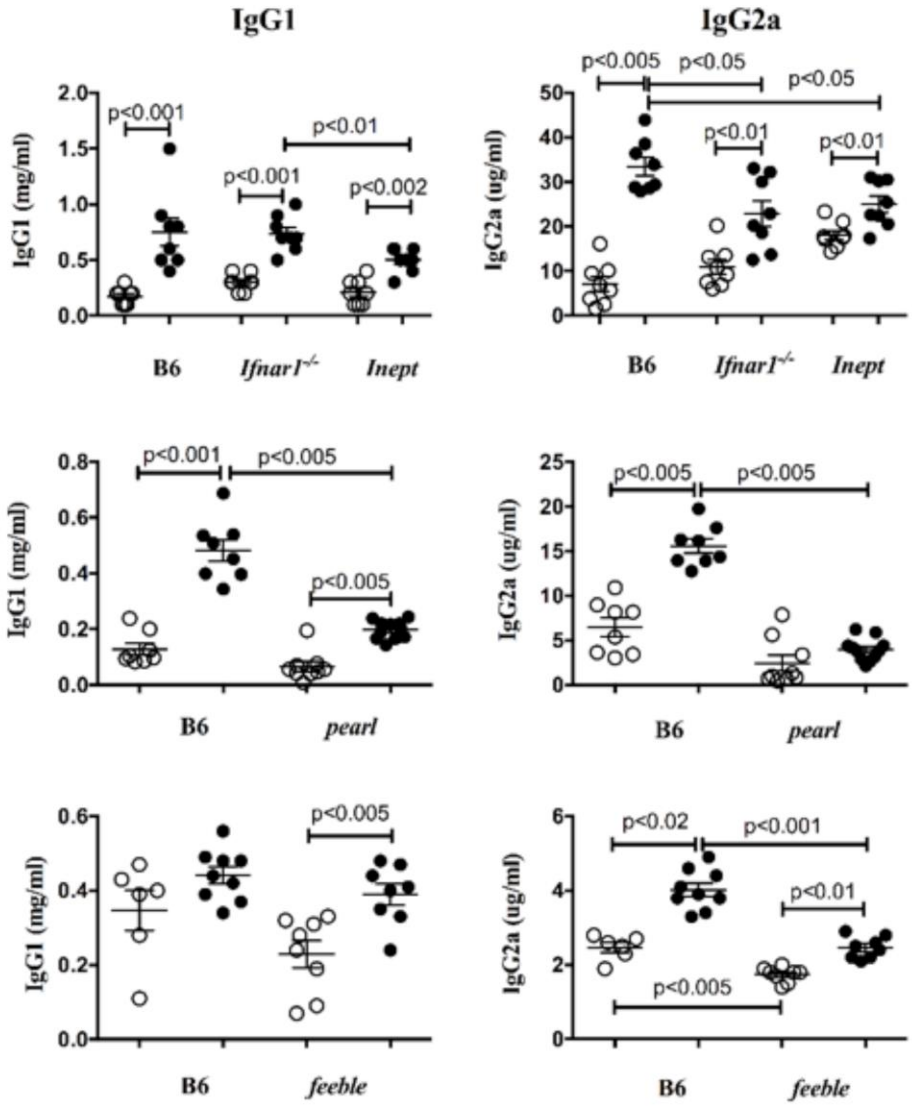
Supplemental Figure 2



Supplemental Figure 2. IgG1 and IgG2c(a) anti-chromatin autoantibodies in mutant and wild type B6 mice after exposure to $HgCl_2$.

Dot plots of serum of IgG1 and IgG2c anti-chromatin autoantibodies in mice deficient for type I IFN receptor (*Ifnar1-/-*), IRF-7 (Irf7-/-), or AP3B1 (*Ap3b1-/-*) after a 4-week exposure to 40 μg $HgCl_2$ s.c. twice-a-week. The corresponding B6 (C57BL/6) wild type control was included for each assay. Anti-DNA and -ENA5 levels were measured by ELISA. $P<0.05$ are shown.

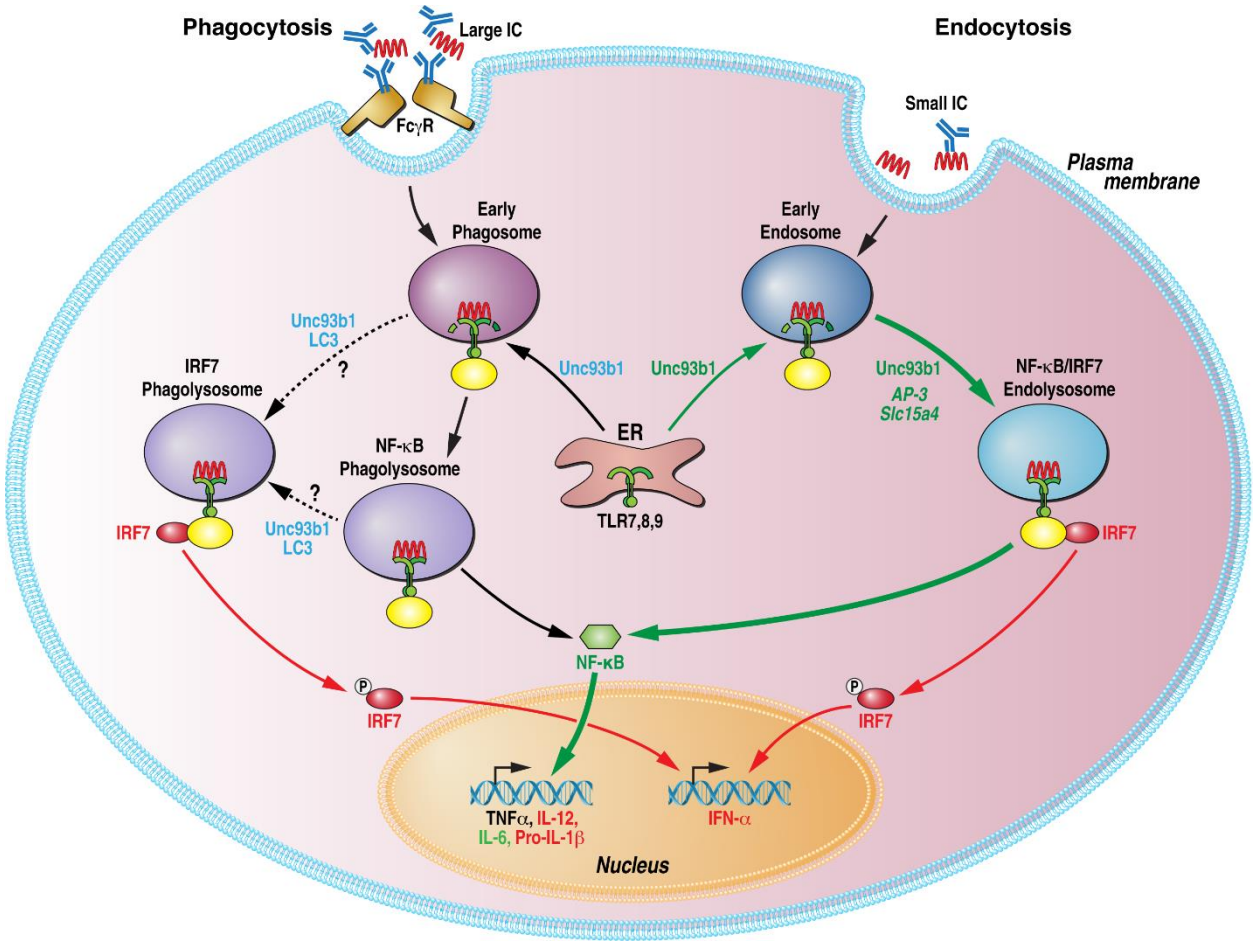
Supplemental Figure 3



Supplemental Figure 3. IgG1 and IgG2a subclasses in mutant and wild type mice after exposure to $HgCl_2$.

Dot plots of serum IgG subclasses in mice deficient for type I IFN receptor (*Ifnar1*$^{-/-}$) and IRF-7 (*Inept*), after a 4-week exposure to 40 μg $HgCl_2$ s.c. twice-a-week. The corresponding wild type controls are included for each mutant. IgG1 and IgG2a levels were measured by ELISA. P<0.05 are shown.

Supplemental Figure 4



Supplemental Figure 4. TLR-related endosomal pathways implicated in type I IFN-independent HgIA.
Phagolysosomal and endolysosomal compartments and pathways associated with nucleic acid-sensing TLR signaling are depicted. Endosomal trafficking (UNC93B1, AP-3), proton solute-transport (SLC15A4), or TLR-signaling (NF-κB) proteins and related pathways shown in this study to be required for type I IFN-independent HgIA are indicated in green. Tentative pathways (dotted arrows) are indicated. A single AP-3-dependent endosome/lysosome compartment activating both NF-κB and IRF7 signaling is depicted, however, distinct signaling compartments are also possible.