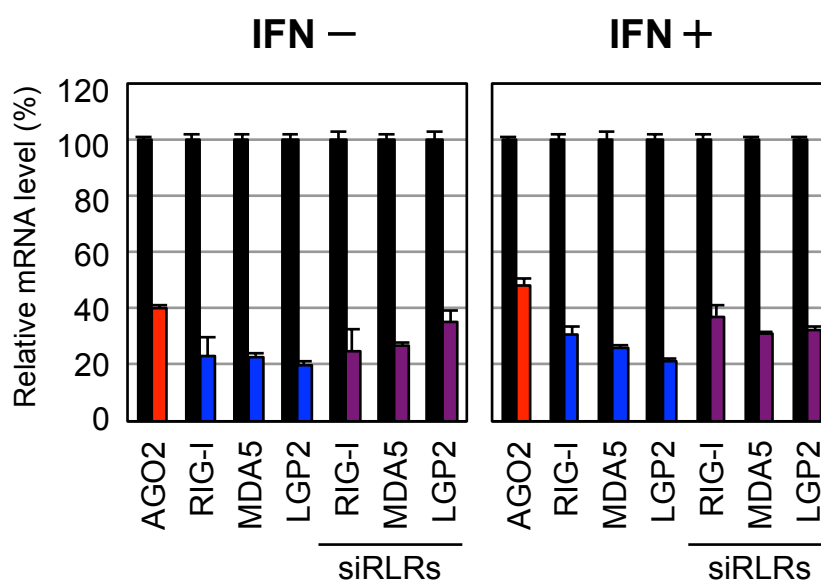


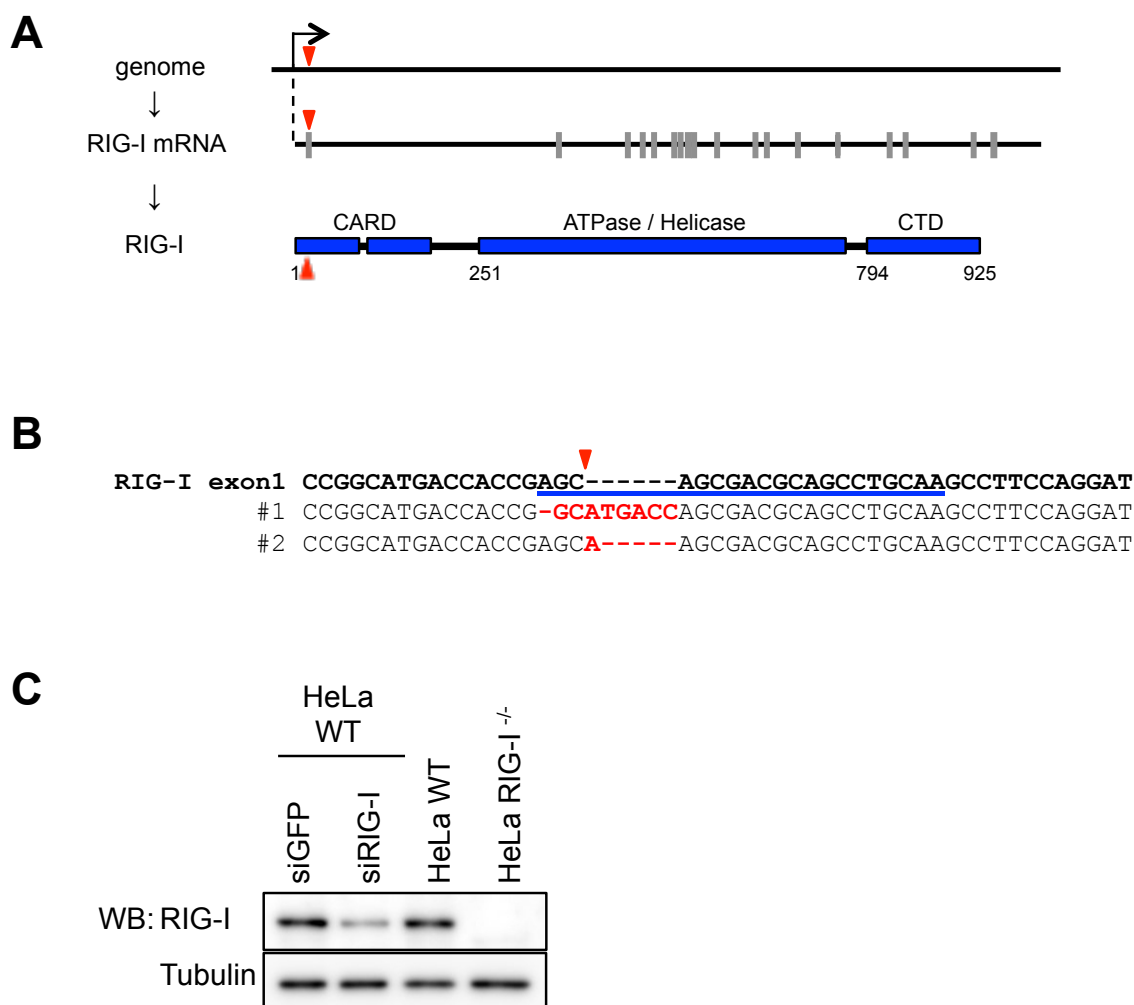
Supplementary Materials for

Virus Sensor RIG-I represses RNA interference by interacting with TRBP through LGP2 in mammalian cells

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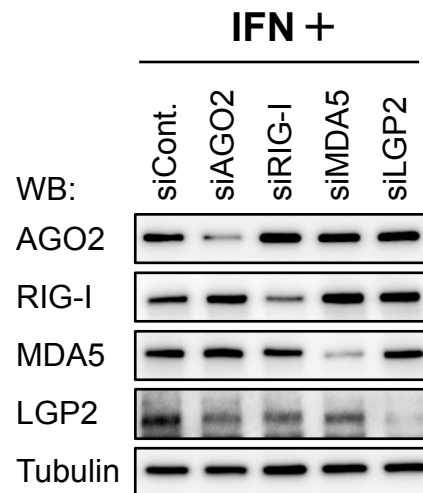


Supplementary Figure 1. Relative mRNA level of AGO2, RIG-I, MDA5, or LGP2 in IFN-non treated or treated cells. The mRNA level of each gene in IFN-non treated or treated cells was measured by quantitative RT-PCR. Change in the expression level of each mRNA (red, blue, purple bar) was normalized with GAPDH mRNA (black bar) in the same cells, and the relative mRNA level in IFN-non treated or treated cells was shown by normalization using the value in the control cells.



Supplementary Figure 2. Generation of RIG-I knockout HeLa cells (RIG-I^{-/-}).

(A) The sgRNA was designed against exon 1 of genomic RIG-I gene. Gray box and arrowhead indicate exons and the predicted cleavage site by Cas9 protein, respectively. (B) Sequence of RIG-I gene in the cloned RIG-I^{-/-} cells. Two patterns of mutation were detected. Blue line indicates the complementary region of the used guide RNA. (C) Western blot was performed using IFN-treated wild-type HeLa cells transfected with siGFP or siRIG-I, and wild-type HeLa cells and RIG-I^{-/-} cells.



Supplementary Figure 3. Confirmation of knockdown at protein level by transfection of siAGO2, siRIG-I, siMDA5, or siLGP2 in TRBP^{-/-} cells.

Western blot of IFN-treated TRBP^{-/-} cells after transfection of each siRNA against AGO2, RIG-I, MDA5, or LGP2. The protein level derived from the knocked down mRNA was sufficiently downregulated.