Supplementary Figure Legends

Supplementary Figure 1. All primary MEFs utilized in this study are equally susceptible to VSVgfp infection. Untreated MEFs were infected with VSVgfp at an moi of 0.1 and raw fluorescence units (RFUs) were measured 24hr post-infection. There were no statistically significant differences in infection rates between cells, as measured by a Oneway ANOVA and a Tukey post hoc test ($p=0.0821$). These data are an average of at least three independent experiments per cell type.

Supplementary Figure 2. Cell association and entry are not dependent on dsRNA length. WT WEFs were treated with Alexafluor 488 labeled v200 and v1000 (3nM) for 30 minutes, after which total, cell associated and intracellular fluorescence was measured using a fluorescence plate reader. Intracellular fluorescence was determined following 0.025% trypan blue treatment to quench surface bound fluorescent dsRNA. The y-axis represents cell associated and intracellular fluorescence expressed as a percent of the total fluorescence added to each well. These data represent the average of three independent experiments. There was no significant difference (ns) in cell associated and intracellular dsRNA between lengths as determined by one-way ANOVA with a Tukey post test.

Supplementary Figure 3. Induction of IRFs, IFNs and ISGs in response to dsRNA of different lengths. In vitro transcribed dsRNA of various lengths was used to treat WT, IRF3$^{-/-}$ and IRF3/9$^{-/-}$ MEFs using an amount of dsRNA shown to elicit a complete antiviral response in each cell type. RNA harvested 6 hours post treatment was reverse transcribed and the cDNAs were assayed using Mouse Interferons and Receptors RT^{2}Profiler^{TM} PCR Arrays. For details please refer to Figure 3 of the main text.

Supplementary Figure 4. Treatment of MEFs with polyI:C inhibits replication of HSV-1. WT, IRF3$^{-/-}$ and IRF3/9$^{-/-}$ MEFs were treated with polyI:C (1.5nM, 3 nM and 8.5nM respectively) for 6h, followed by infection with HSV-1gfp (moi 0.1) for 24h. The replication of HSV-1 in untreated cells was set to 100% in each cell type. A statistically significant difference

in HSV-1 replication was observed between untreated and polyI:C treated MEFs. All cell types were equally susceptible to infection with HSV-1 (data not shown). *** $p<0.001$.