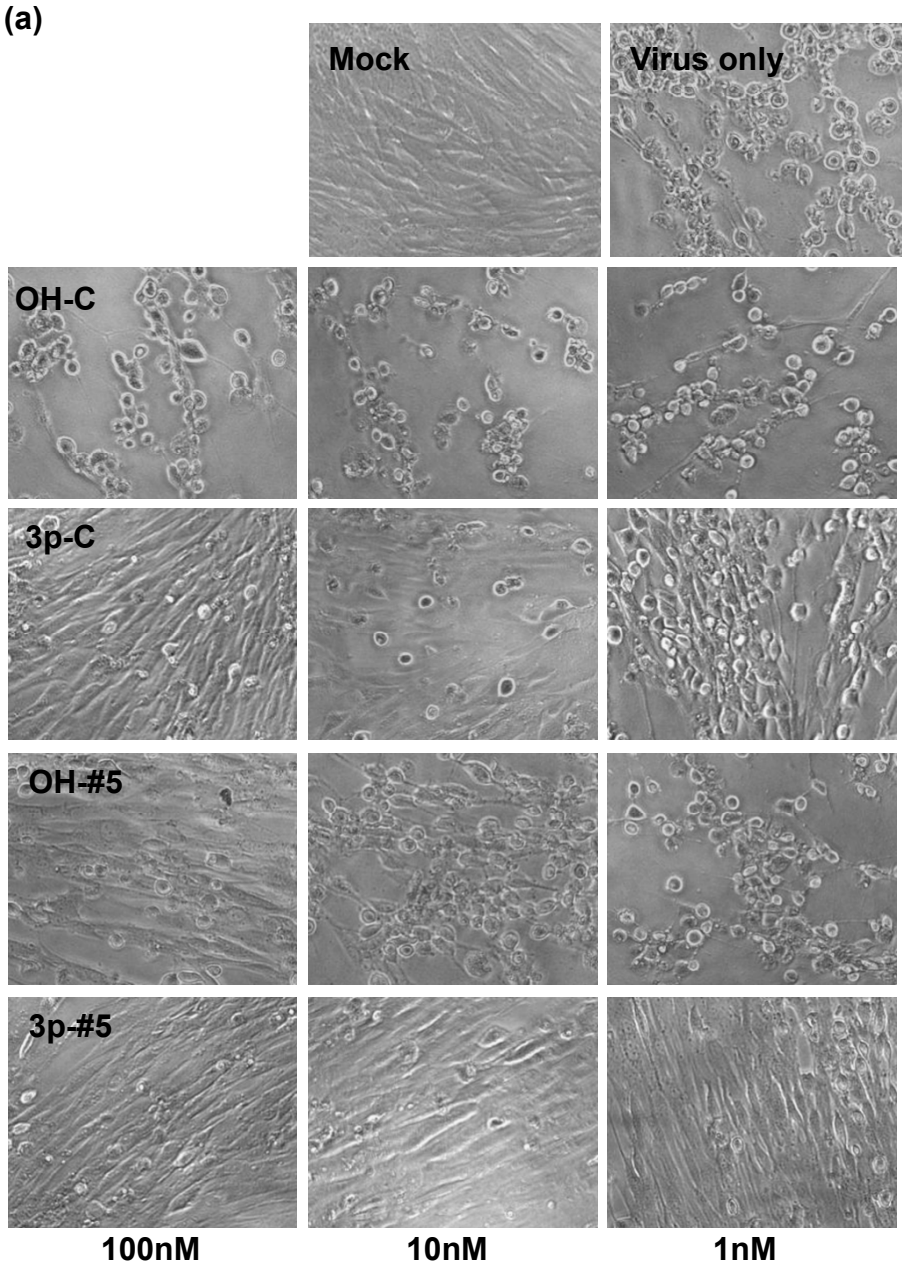
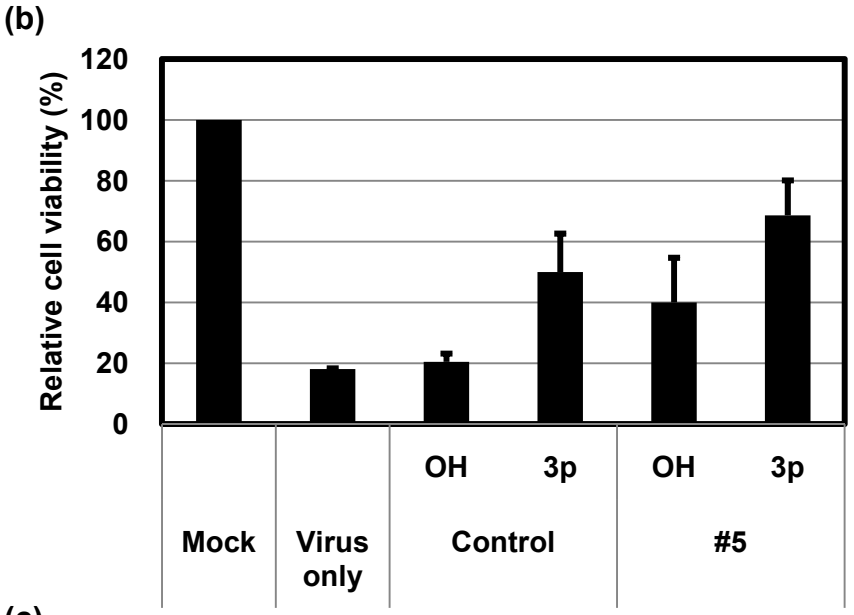
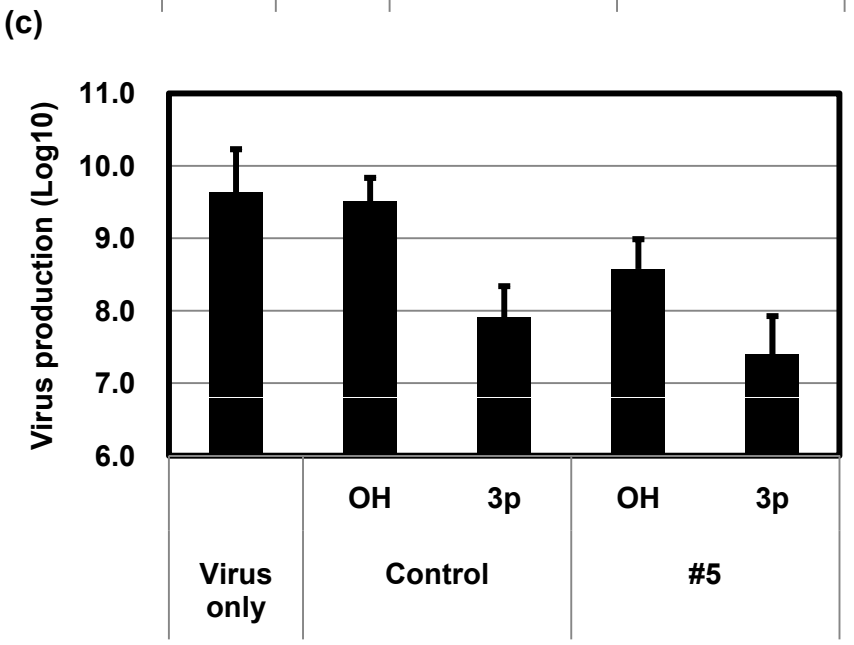
Supple. Fig. 1

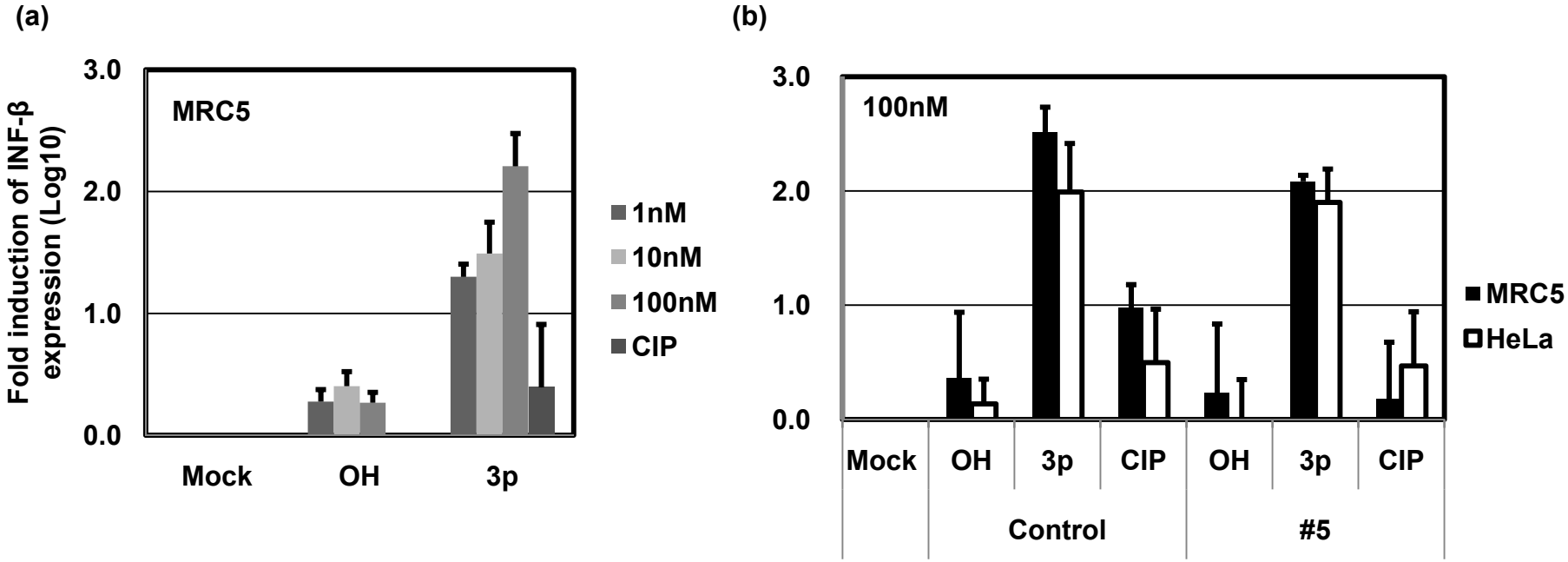

(a)
Mock
Virus only
OH-C
3p-C
OH-#5
3p-#5
100nM
10nM
1nM


(b)
Relative cell viability (%)
120
100
80
60
40
20
0
OH
3p
OH
3p
Mock
Virus only
Control
#5


(c)
Virus production (Log10)
11.0
10.0
9.0
8.0
7.0
6.0
OH
3p
OH
3p
Virus only
Control
#5

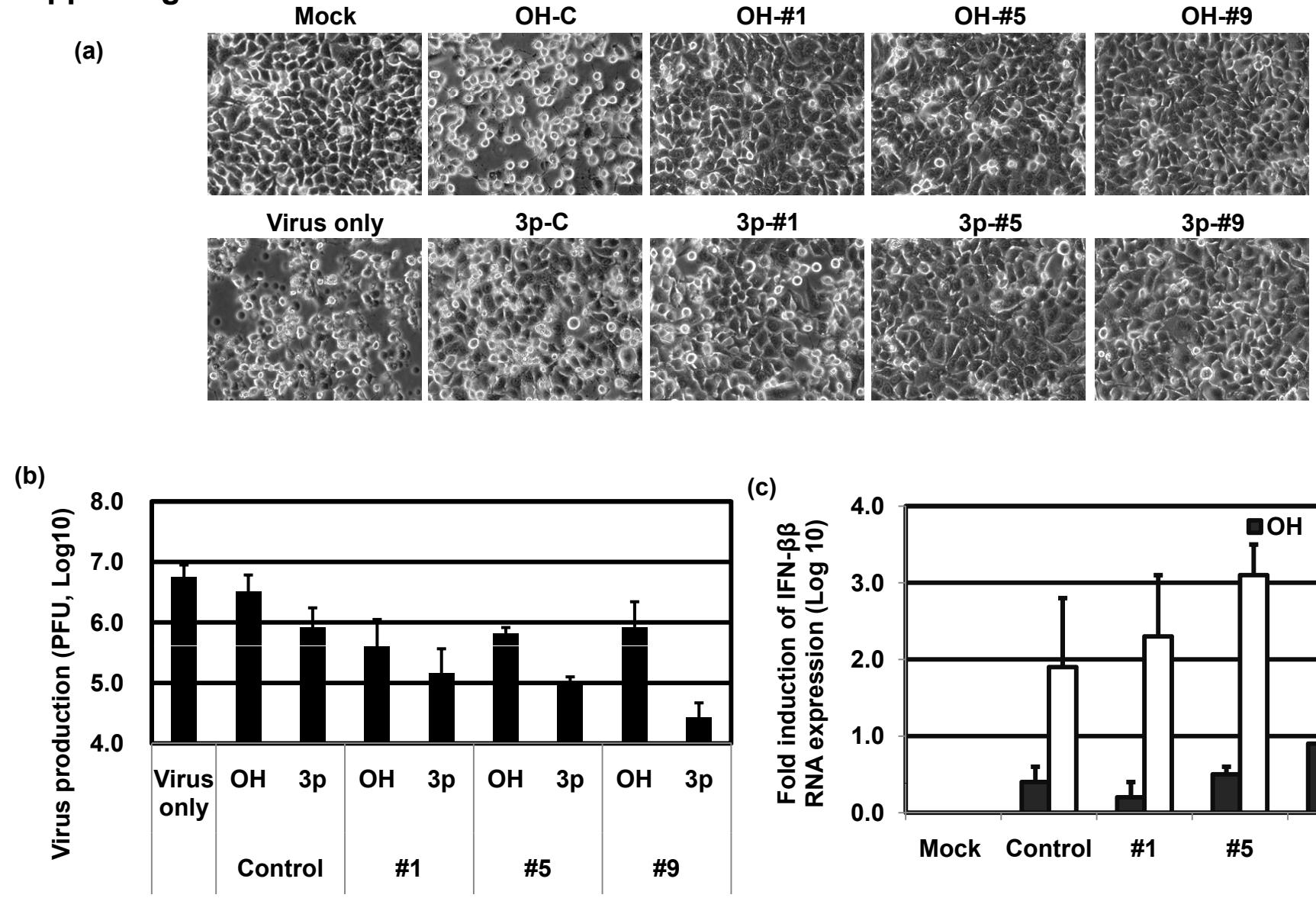
Supplementary figure 1. Protection effects of CVB3-specific 3p-siRNA in MRC5 cells. Cells were infected with CVB3 at MOI=1 for 1 hour in the presence or absence of various types of siRNAs at 1, 10, or 100 nM (n=3). After washing the virus inoculum, the cells were cultured for 24 hours. Cytopathic effects were observed under light microscopy (a; the representatives of the independent studies). Relative cell viability and the degree of virus replication were determined at 10 nM by MTT assay (b) and $TCID_{50}$ (c), respectively. The mean ± SD.

Supple. Fig. 2


(a)
MRC5
Fold induction of INF-β expression (Log10)
3.0
2.0
1.0
0.0
Mock
OH
3p
1nM
10nM
100nM
CIP
(b)
100nM
3.0
2.0
1.0
0.0
Mock
OH
3p
CIP
OH
3p
CIP
Control
#5
MRC5
HeLa

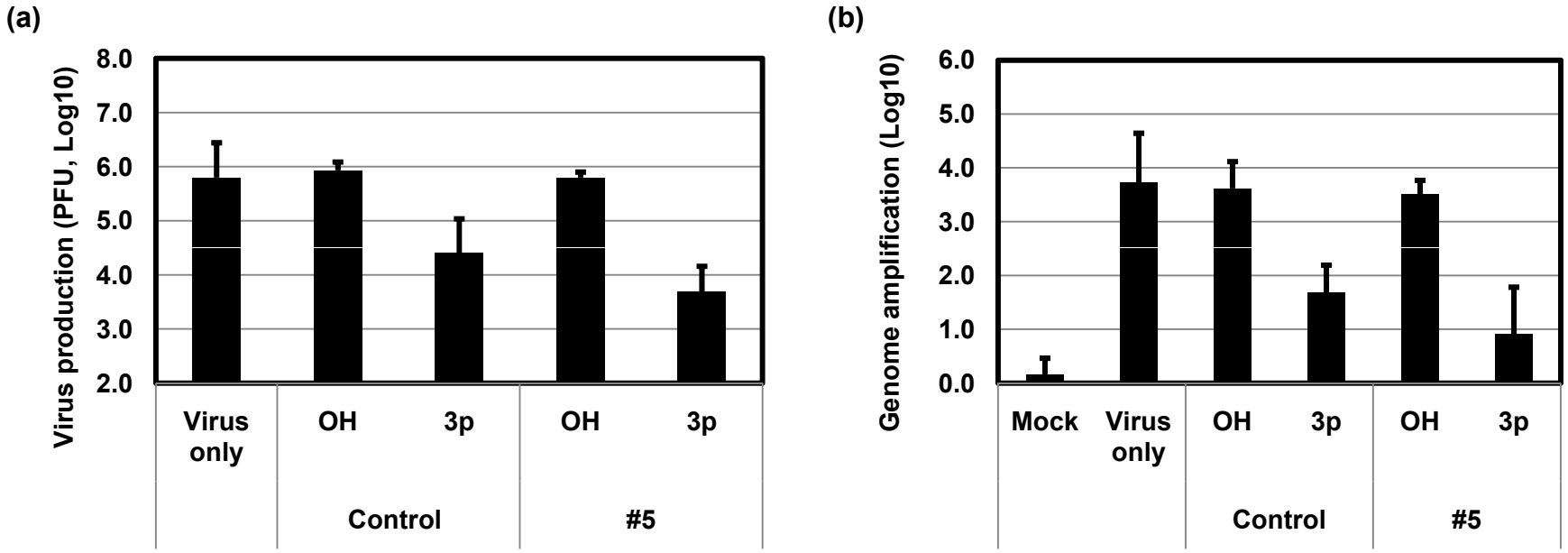
Supplementary figure 2. Induction of INF-β expression following siRNA treatment in cells. Either MRC5 or HeLa cells were treated with various types of siRNAs at 1, 10, 100 nM for 24 hours. Real time RT-PCR was carried out to quntify INF-β mRNA expression. Beta actin was used for normalization in the estimation of relative gene expression. Fold induction of INF-β expression in MRC5 cells at different concentration of #5 (a) and in the cells at 100 nM (b) were shown (n=3). Beta actin was used for normalization in the estimati on of relative gene expression. CIP; calf intestine phosphatase-treated siRNAs (100 nM). The mean ± SD.

Supple. Fig. 3


(a)
Mock
OH-C
OH-#1
OH-#5
OH-#9
Virus only
3p-C
3p-#1
3p-#5
3p-#9
(b)
Virus production (PFU, Log10)
8.0
7.0
6.0
5.0
4.0
Virus only
OH
3p
Control
#1
#5
#9
(c)
Fold induction of IFN-ββ RNA expression (Log 10)
4.0
3.0
2.0
1.0
0.0
OH
3p
Mock
Control
#1
#5
#9

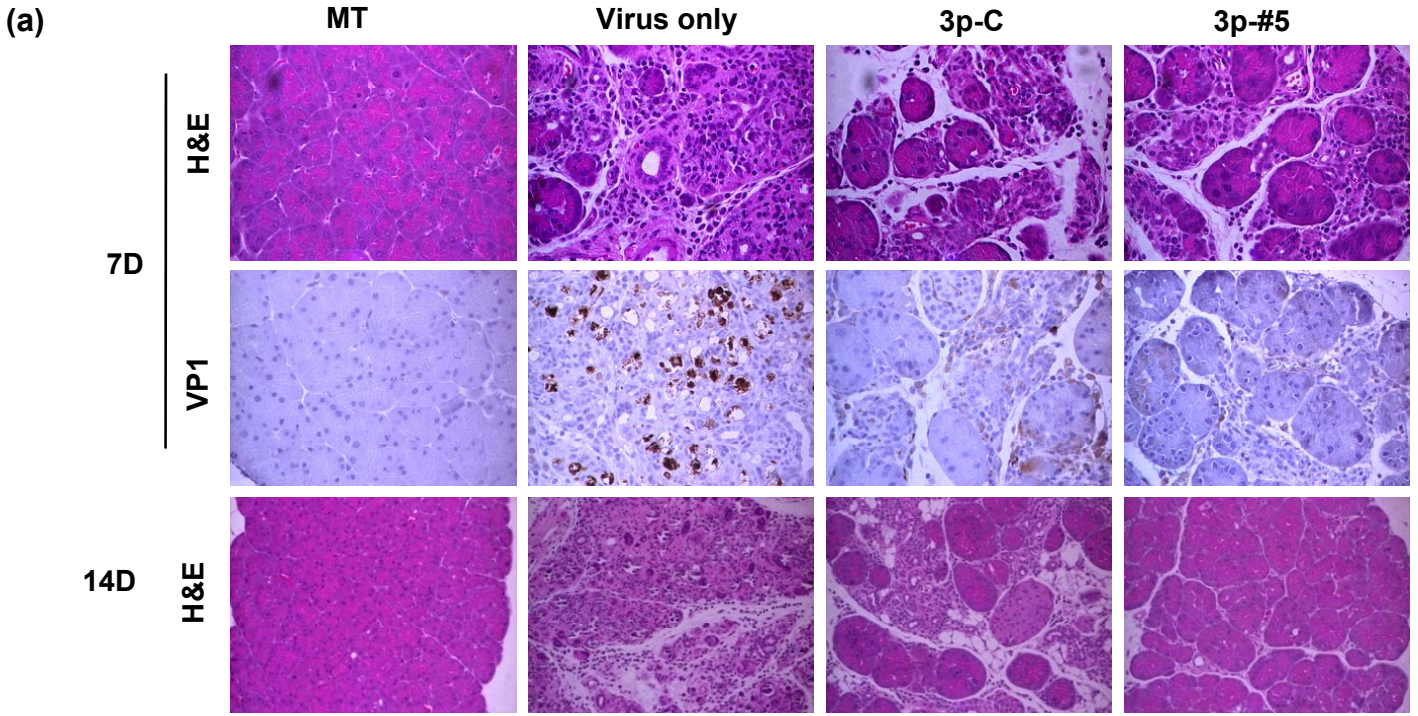
Supplementary figure 3. Characteristics of various siRNAs *in vitro*. HeLa cells were preincubated with various types of siRNAs at 100 nM for 15 hours, followed by CVB3 infection at MOI=1. Cytopathic effects were observed under light microscopy at 10 h pi (a, the representatives of 3 independent studies). Cells and culture media were collected at 8 h pi and progeny virus production was determined by $TCID_{50}$ (b, n=4). To quantify INF-β mRNA expression, MRC5 cells were treated with various types of siRNAs at 10 nM for 24 hours (c, n=3). Real time RT-PCR was carried out and beta actin was used for normalization in the estimation of relative gene expression. The mean ± SD.

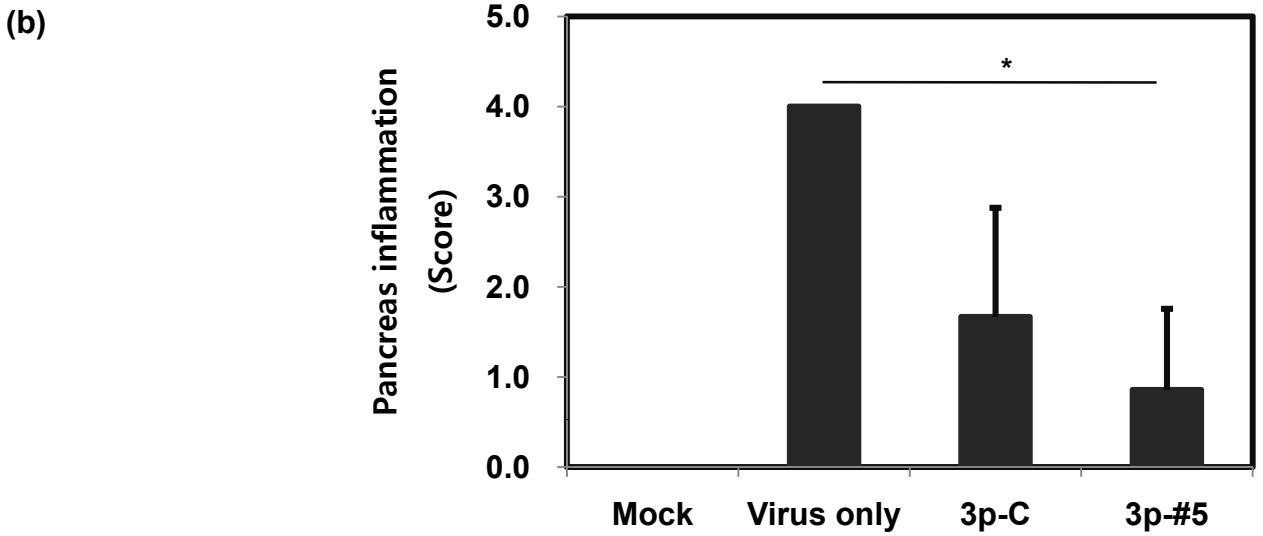
Supple. Fig. 4


(a)
Virus production (PFU, Log10)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
Virus only
OH
3p
OH
3p
Control
#5
(b)
Genome amplification (Log10)
6.0
5.0
4.0
3.0
2.0
1.0
0.0
Mock
Virus only
OH
3p
OH
3p
Control
#5

Supplementary figure 4. Virus replication in heart tissues following various siRNA treatments. Mice were injected intraperitoneally with 8×10^5 PFU/200μl of virus and intravenously administered with 2.5 mg/kg of various siRNAs per mouse after 8 h. Progeny virus production was tested by $TCID_{50}$ on day 7 (a, n=3 for each group). The viral genome was quantified on day 14 using RT-PCR (b, n≥4 for each group). The mean ± SD.

Supple. Fig. 5


(a)
MT
Virus only
3p-C
3p-#5
7D
H&E
VP1
14D
H&E


(b)
Pancreas inflammation
(Score)
5.0
4.0
3.0
2.0
1.0
0.0
*
Mock
Virus only
3p-C
3p-#5

Supplementary figure 5. Histopathological examination of pancreas following virus and siRNA treatment. Pancreases were recovered and paraffin blocks were prepared for histopathological analysis, as described in Fig. 4 (n=6). Sections were stained with H&E and immunohistochemically stained using virus-specific VP1 antibody (a). Pancreas inflammation on day 7 was scored on day 7 (b). The mean ± SD. *P<0.05.