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Corrigendum to "3C^{pro} of FMDV inhibits type II interferon-stimulated JAK-STAT signaling pathway by blocking STAT1 nuclear translocation" [Virol Sin 38 (2023) 387–397]

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Due to our negligence, the original version of this article, published online on Mar 14, 2023, contained some mistakes in several Figs.

In Fig. 2D, the positions of the bands for protein 3A and 3B were incorrectly shifted. This has been modified in corrected Fig.2 as shown below.

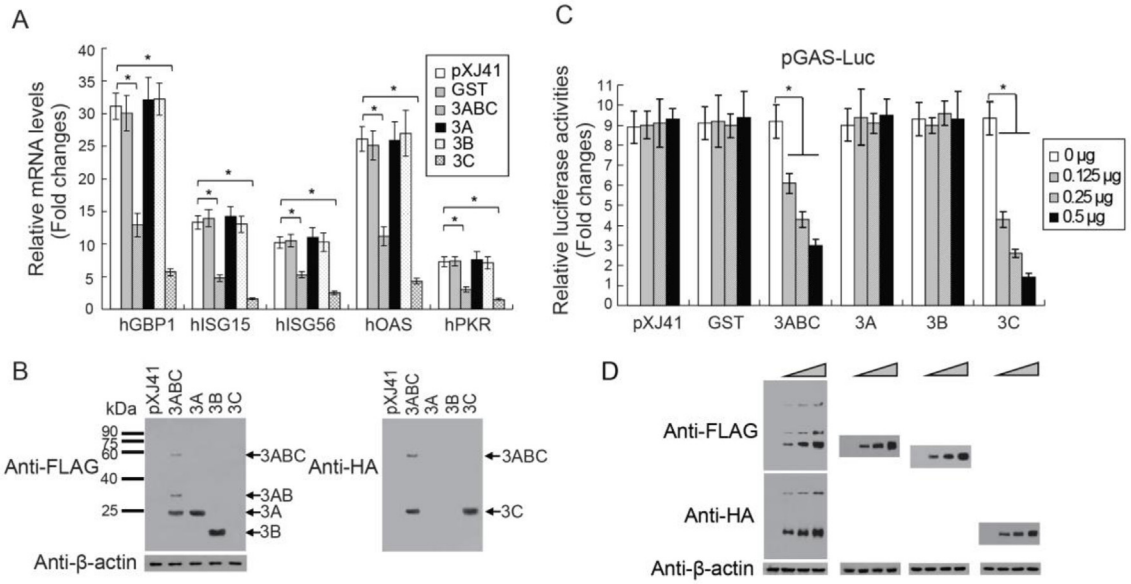

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Fig. 2. FMDV 3C^{pro} suppressed ISGs mRNA synthesis and GAS promoter activity of type II IFN signaling. **A** HeLa cells transfected with pXJ41, pXJ41-GST, pXJ41-3ABC, pXJ41-3A, pXJ41-3B or pXJ41-3C were treated with 100 ng/mL of human IFN-γ at 24 h after transfection. 5 h later, the ISGs transcript levels were measured by real-time RT-PCR, with pXJ41 and pXJ41-GST as negative controls. The levels of relative transcript were shown as relative fold changes, compared with the mock-treated control level of uninfected cells. Data represent the mean ± the standard deviations (error bars) of three independent experiments. **B** Expression of 3ABC, 3A, 3B or 3C protein was detected by Western blotting using antibody against FLAG or antibody against HA. β-actin was used as a protein loading control. **C** HeLa cells grown in 12-well plates were cotransfected with 0 μg, 0.125 μg, 0.25 μg or 0.5 μg of pXJ41, pXJ41-GST, pXJ41-3ABC, pXJ41-3A, pXJ41-3B or pXJ41-3C and 0.5 μg of pGAS-Luc as well as 0.05 μg of pRL-TK as an internal control. pXJ41 and pXJ41-GST were worked as negative controls. At 24 h after transfection, cells were treated with 100 ng/mL of human IFN-γ. 16 h later, cells were lysed and reporter expressions were measured using the Dual-Luciferase reporter assay kit (Promega). The values were normalized regarding *Renilla* luciferase activities. Then relative expression levels were calculated and shown as relative fold changes, compared with the mock-treated control of untransfected cells. Data represent the mean ± the standard deviations (error bars) of three independent experiments. **D** Expression of 3ABC, 3A, 3B or 3C protein was detected by Western blotting using antibody against FLAG or antibody against HA. β-actin was used as a protein loading control. *, $P < 0.05$.

In Fig. 3A, the vertical axis marking of Figure 3A was incorrect in previous version. The correct Fig.3 is given below.

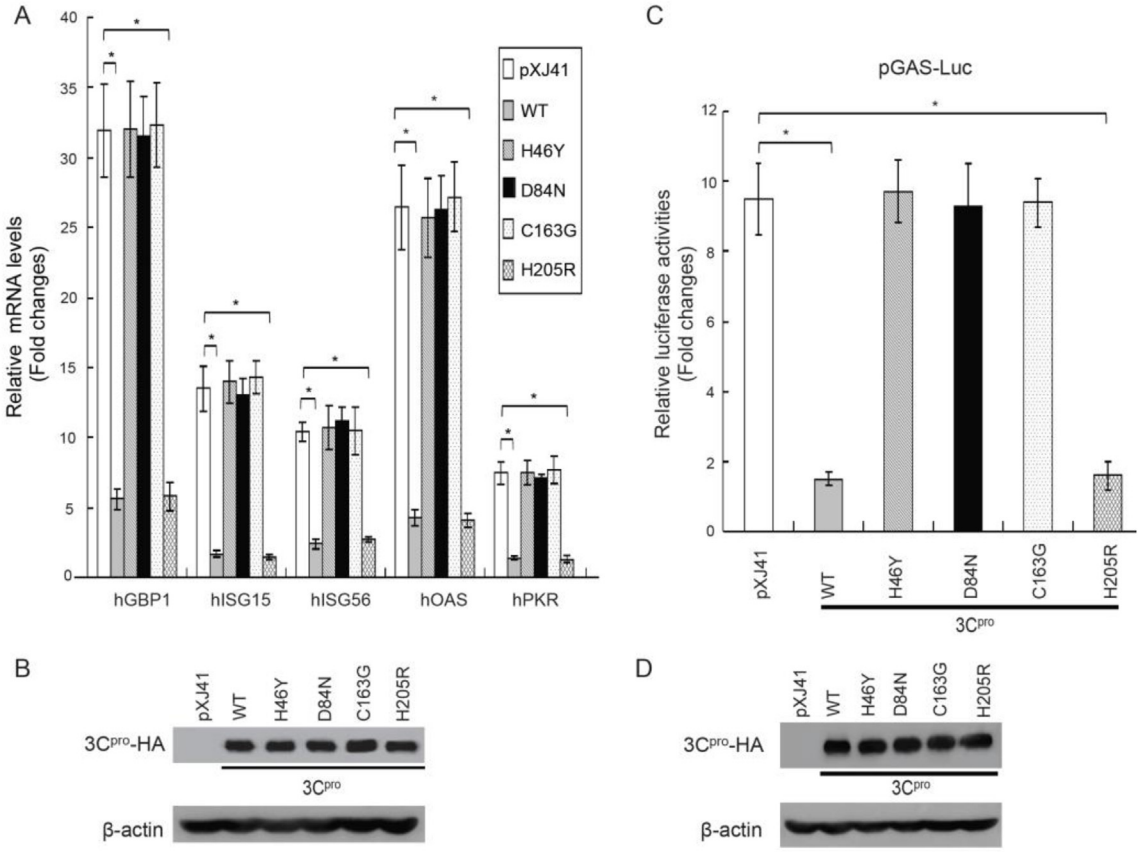

Fig. 3. The catalytic triad H46, D84 and C163 of 3C^{pro} played essential roles in suppressing ISGs mRNA synthesis and GAS promoter activity of type II IFN signaling. **A** HeLa cells transfected with pXJ41, pXJ41-3C or individual mutants of pXJ41-3C were treated with 100 ng/mL human IFN-γ at 24 h after transfection. 5 h later, the hGBP1, hISG15, hISG56, hOAS and hPKR transcript levels were analyzed by real-time RT-PCR as described above. Data represent the means ± the standard deviations (error bars) of three independent experiments, with each experiment in triplicate. **B** Expression of 3C or individual mutant protein was detected by Western blotting using antibody against HA. β-actin was used as a protein loading control. **C** HeLa cells transfected with 0.5 μg of pXJ41, pXJ41-3C or its individual mutants of pXJ41-3C and 0.5 μg of pGAS-Luc as well as 0.05 μg of pRL-TK. At 24 h after transfection, cells were treated with 100 ng/mL human IFN-γ. 16 h later, cells were lysed and reporter expressions were measured using the Dual-Luciferase reporter assay kit as described above. Data represent the mean ± the standard deviations (error bars) of three independent experiments. **D** Expression of 3C or individual mutant protein in HeLa cells transfected with indicated plasmid was detected by Western blotting using antibody against HA. β-actin was used as a protein loading control. *, $P < 0.05$.

In Fig. 4A-D, IFN-β should be IFN-γ, it was mislabeled in previous version. In Fig 4D, the legend "Dimer of STAT1" was omitted during the editing process. The correct Fig.4 is given below.

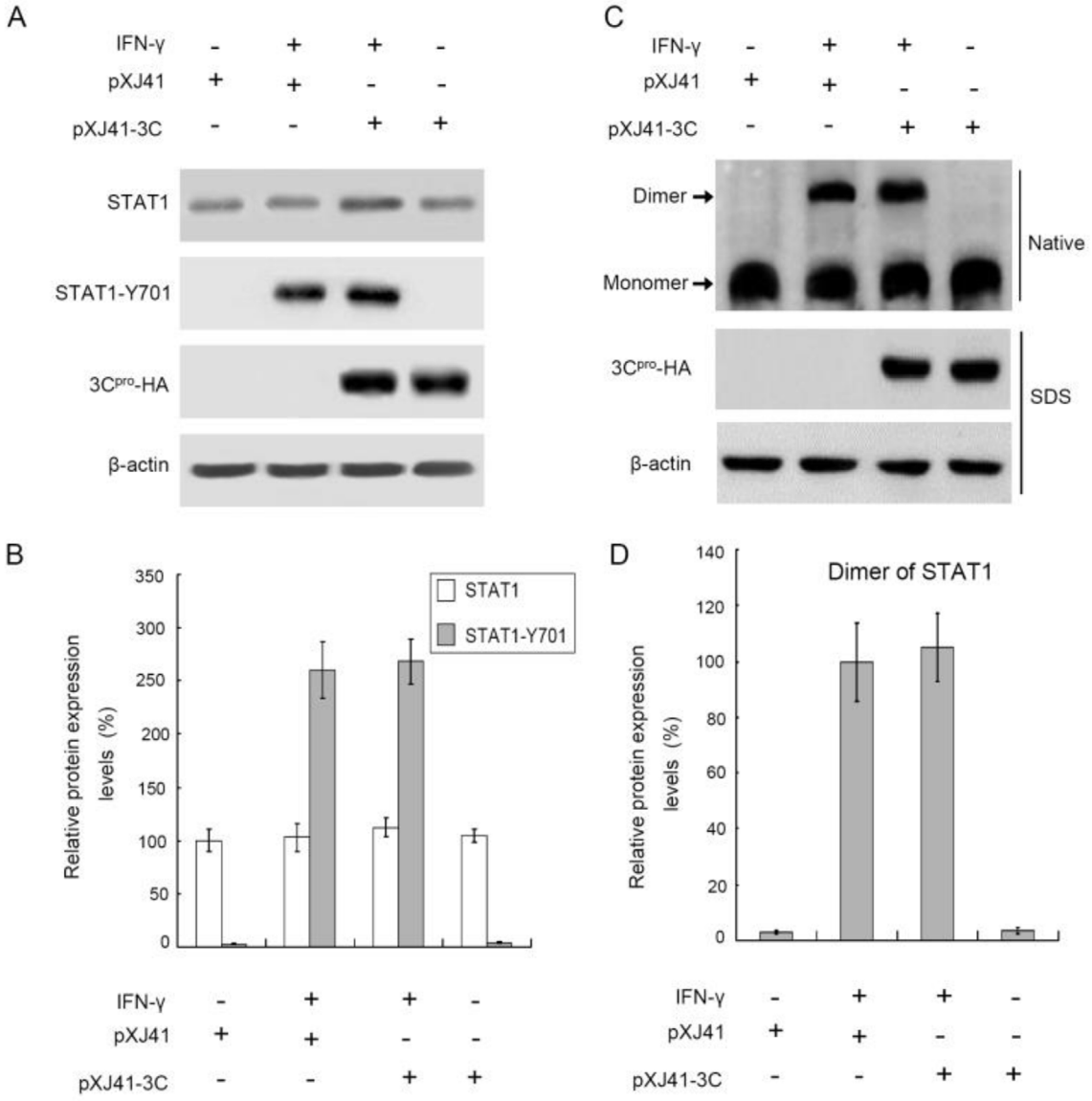

Fig. 4. STAT1 expression level, phosphorylation status and its homodimer formation in FMDV $3C^{pro}$-expressing cells after IFN-γ stimulation. **A** HeLa cells transfected with pXJ41 or pXJ41-3C were treated with 100 ng/ml human IFN-γ for 1 h at 24 h after transfection and collected for Western blotting with antibody against STAT1 (top panel), phospho-STAT1 (STAT1-Y701, panel 2) or HA (panel 3), respectively. Antibody against β-actin (bottom panel) was used as a protein loading control. **B** Densitometric analysis of the digital image in **A**. Intensities of the band were normalized with that of β-actin. **C** Cells transfected with pXJ41 or pXJ41-3C were treated with human 100 ng/mL IFN-γ for 1 h at 24 h post-transfection. Samples were lysed and subjected to native-PAGE and immunoblotting with antibody against STAT1 (top panel). Expression of $3C^{pro}$ was detected by SDS-PAGE and immunoblotting with antibody against HA (panel 2), with β-actin as a loading control (bottom panel). **D** Densitometric analysis of the digital image in **C**. Intensities of the band were normalized with that of β-actin. Data represent the mean ± the standard deviations (error bars) of three independent experiments.

In Fig. 8A-E, there was a discrepancy between the fill color and pattern of the columns and the graphic labels within the legend box. In Fig. 8F, the bands of β-actin in WT, H46Y, and H205R groups were missing. The correct Fig.8 is given below.

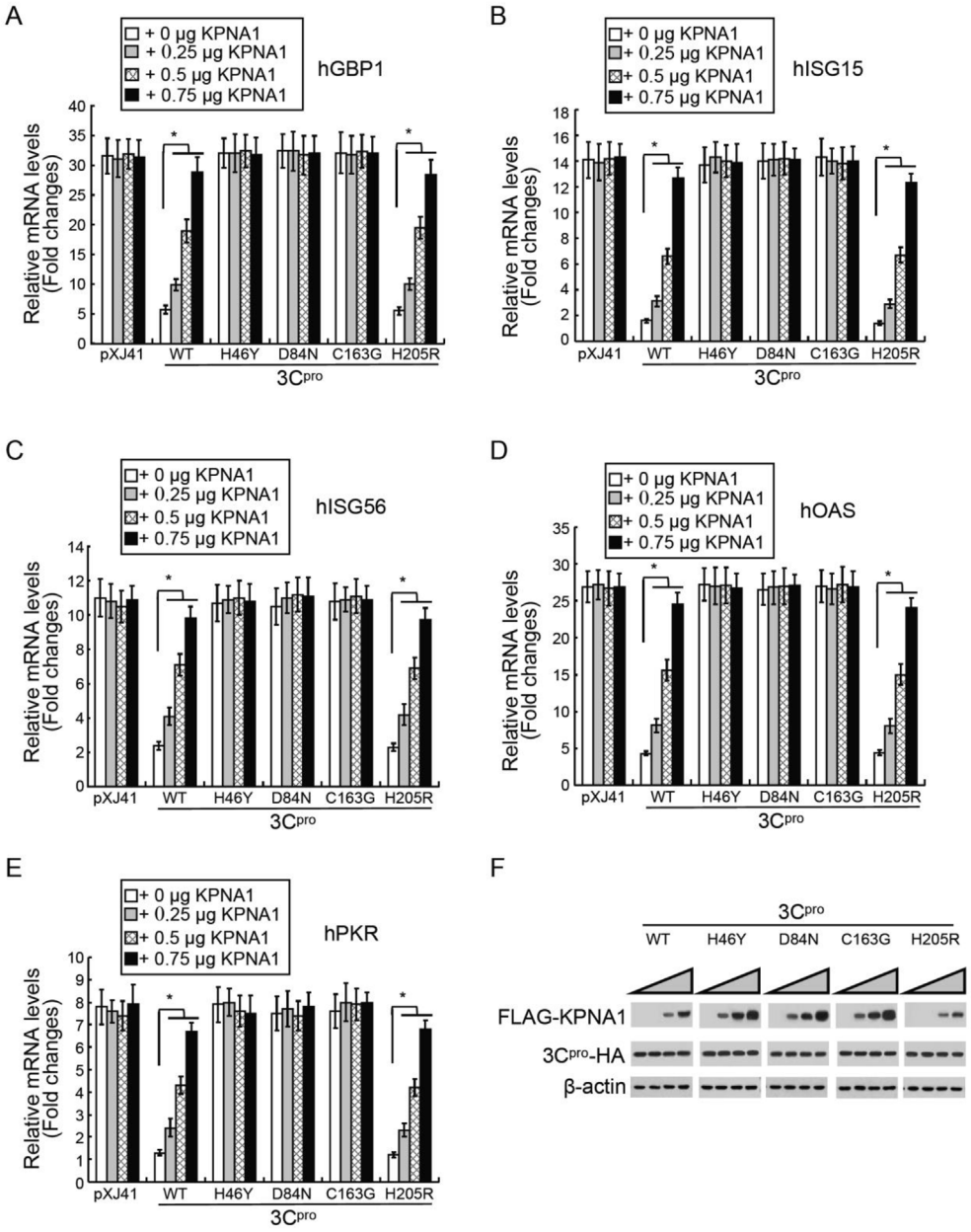

Fig. 8. ISGs mRNA level of type II IFN signaling is associated with FMDV 3C protease activity in degrading KPNA1. HeLa cells cotransfected with increased dose of pXJ41-FLAG-KPNA1 and pXJ41-3C or its mutants were treated with 100 μg/mL human IFN-γ at 24 h post-transfection. 5 h later, the mRNA levels of hGBP1 **(A)**, hISG15 **(B)**, hISG56 **(C)**, hOAS **(D)** and hPKR **(E)** were detected by real-time RT-PCR as described above. Data represent the mean ± the standard deviations (error bars) of three independent experiments. **F** Expression of KPNA1, 3C or its mutant protein was measured by Western blotting using antibody against FLAG or HA. β-actin was used as a protein loading control. *, $P < 0.05$.

We apologize for our oversight when preparing the figures and state that this does not change the scientific conclusions of the article in any way.