CORRECTION



Correction: Aloe-emodin inhibits African swine fever virus replication by promoting apoptosis via regulating NF-kB signaling pathway

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In this article [1], Fig(s) 4 and 5 appeared incorrectly and have now been corrected in the original publication. For completeness and transparency, both the incorrect and correct versions are displayed below.

The original article can be found online at https://doi.org/10.1186/s12985-023-02126-8.

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Incorrect Figs. 4 and 5



Fig. 4 Ae inhibits the NF- κ B signaling pathway activated by ASFV infection. The expression level of (**A**) MyD88 protein, (**B**) phospho-NF- κ B p65 protein, and (**C**) pl κ B protein in the ASFV-infected group, ASFV-infected group treated with BAY11-7082 or Ae was detected by western blot. The expression of tubulin was used as a positive control. qPCR was used to detect the changes of (**D**) IL-1 β mRNA levels and (**E**) IL-8 mRNA levels in the ASFV-infected group treated with BAY11-7082, or Ae at different time points. The mRNA level of GAPDH was used as a positive control. All control cells were normal cultured PAMs. The results of three independent experiments (mean ± SD) were represented by one data. Significant differences that were compared to the control group were indicated by * (P < 0.05), ** (P < 0.01) and *** (P < 0.001)



Fig. 5 Ae promotes apoptosis by inhibiting the NF-κB signaling pathway. (**A**) The apoptosis of PAMs in the ASFV infection group, BAY11-7082 or Ae-treated ASFV infection group was detected by flow cytometry, and the apoptosis of induced cells was detected at 3, 12 and 48 h after treatment. Untreated cells served as negative controls. The expression level of (**B**) Bcl-2 protein, (**C**) cleaved-caspase3 protein, and (**D**) Bax protein in the ASFV infection group, BAY11-7082 or Ae-treated ASFV infection group was detected by western blot. All control cells were normal cultured PAMs. The tubulin expression was used as a positive control



Correct Figs. 4 and 5

Fig. 4 Ae inhibits the NF- κ B signaling pathway activated by ASFV infection. **A** The expression level of MyD88 protein, phospho-NF- κ B p65 protein, and pl κ B protein in the ASFV-infected group, ASFV-infected group treated with BAY11-7082 or Ae was detected by western blot. The expression of tubulin was used as a positive control. qPCR was used to detect the changes of **B** IL-1 β mRNA levels and **C** IL-8 mRNA levels in the ASFV-infected group, ASFV-infected group treated with BAY11-7082, or Ae at different time points. The mRNA level of GAPDH was used as a positive control. All control cells were normal cultured PAMs. The results of three independent experiments (mean ± SD) were represented by one data. Significant differences that were compared to the control group were indicated by * (P < 0.05), ** (P < 0.01) and *** (P < 0.001)



Annexin V

Fig. 5 Ae promotes apoptosis by inhibiting the NF-κB signaling pathway. A The apoptosis of PAMs in the ASFV infection group, BAY11-7082 or Ae-treated ASFV infection group was detected by flow cytometry, and the apoptosis of induced cells was detected at 3, 12 and 48 h after treatment. Untreated cells served as negative controls. The expression level of B Bcl-2 protein, cleaved-caspase3 protein, and Bax protein in the ASFV infection group, BAY11-7082 or Ae-treated ASFV infection group was detected by western blot. All control cells were normal cultured PAMs. The tubulin expression was used as a positive control

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