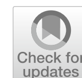
Pharmacological Reports (2024) 76:435–438
https://doi.org/10.1007/s43440-024-00591-8

CORRECTION



Correction: Protective effect of hygrolansamycin C against corticosterone-induced toxicity and oxidative stress-mediated via autophagy and the MAPK signaling pathway

Jongtae Roh[1,2] · Jun-Pil Jang[1] · Taehoon Oh[1,3] · Jihong Kim[1,3] · Byeongsan Lee[1,3] · Young-Soo Hong[1,2] · Jae-Hyuk Jang[1,2] · Sung-Kyun Ko[1,2]

Published online: 4 April 2024
© The Author(s) under exclusive licence to Maj Institute of Pharmacology Polish Academy of Sciences 2024

Correction: Pharmacological Reports https://doi.org/10.1007/s43440-024-00572-x

In this article "μ" symbol was missing from the "μM" notation for substance concentration in Figs. 2 and 4. Additionally, the symbol "β" in "β-Actin" is missing in Figs. 5, 6, 7. The figures should have appeared as shown below.

The original article has been corrected.

The original article can be found online at https://doi.org/10.1007/s43440-024-00572-x.

✉ Jae-Hyuk Jang
jangjh@kribb.re.kr

✉ Sung-Kyun Ko
ksk1230@kribb.re.kr

1 Chemical Biology Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Cheongju 28116, Korea

2 KRIBB School of Bioscience, Korea University of Science and Technology (UST), Daejeon, Korea

3 College of Pharmacy, Chungbuk National University, Cheongju, Korea

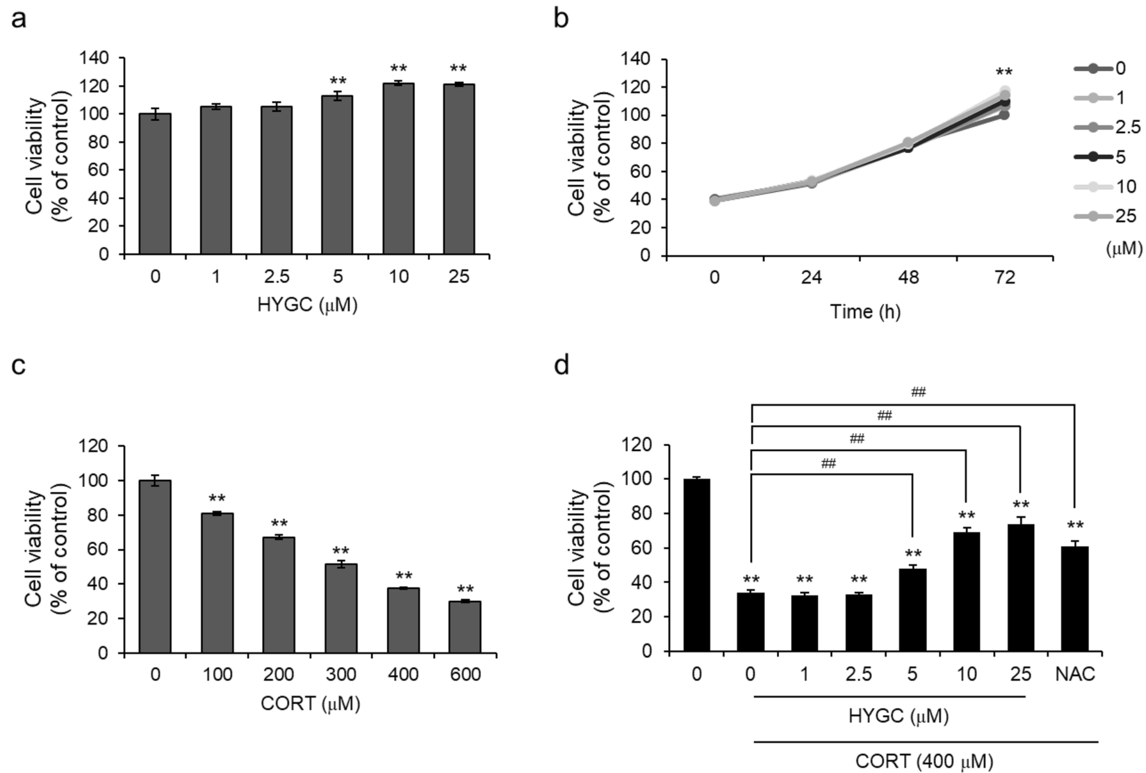


Fig. 2 HYGC inhibits corticosterone (CORT)-induced cytotoxicity in PC12 cells. **a**, **b** PC12 cells were incubated with the indicated concentration of HYGC or DMSO for 24 h or the indicated time. Cell viability was measured using the EZ-Cytox colorimetric kit (mean ± SD, n = 3). **c** PC12 cells were incubated with various concentrations of CORT for 24 h. Cell viability was measured using the EZ-Cytox colorimetric kit (mean ± SD, n = 3). **d** PC12 cells were incubated with the indicated concentration of HYGC in the absence or presence of CORT (400 μM). NAC (5 mM) was used as a positive control. Cell viability was measured using the EZ-Cytox colorimetric kit. Data were analyzed using **a**, **c**, **d** one-way ANOVA followed by Dunnett's post hoc test and **b** two-way ANOVA followed by Tukey's post hoc test. (Mean ± SD, n = 3; $^{*}p < 0.05$ and $^{**}p < 0.01$ compared to the DMSO control group; $^{\#\#}p < 0.01$ compared to the group treated with CORT only)

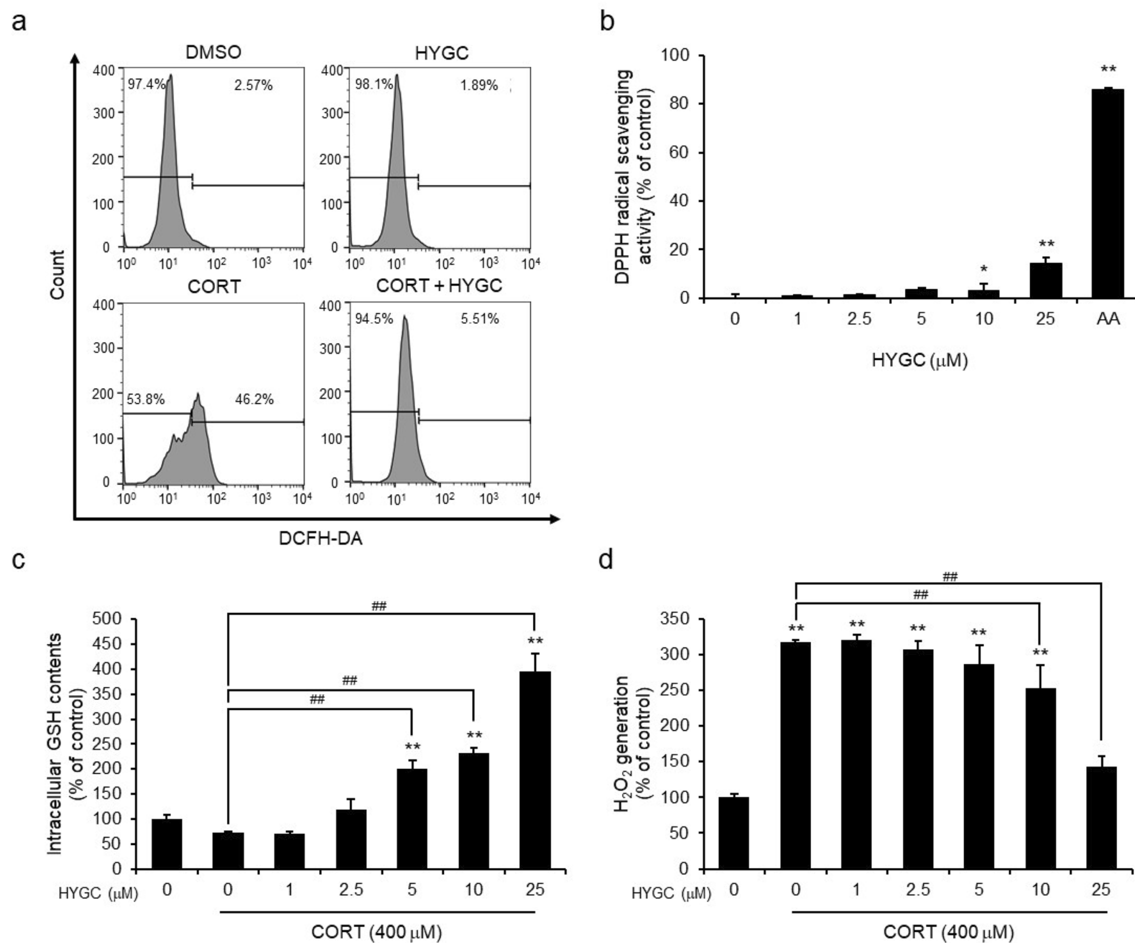


Fig. 4 Inhibitory effects of HYGC on CORT-induced oxidative stress in PC12 cells. **a** PC12 cells were incubated with HYGC (25 μM) in the presence or absence of CORT (400 μM) for 24 h. Cellular ROS production was determined by DCFH-DA assay. The intensity of DCFH-DA was analyzed with flow cytometry. **b** The radical scavenging potential of HYGC was determined using the DPPH assay. AA (1 mM) was used as a positive control (mean ± SD, n = 3). **c**, **d** PC12 cells were incubated with various concentrations of HYGC in the presence or absence of CORT (400 μM) for 24 h. Cultured cells were used to measure intracellular GSH activity, and the supernatant was used to measure H_2O_2 production. **c** Intracellular GSH activity was measured using the GSH-Glo assay kit (mean ± SD, n = 3. **d** H_2O_2 production was measured using the ROS-Glo H_2O_2 assay kit. Data were analyzed using one-way ANOVA followed by Dunnett's post hoc test. (Mean ± SD, n = 3; $*p < 0.05$ and $**p < 0.01$ compared to the DMSO control group; $^{##}p < 0.01$ compared to the group treated with CORT only)

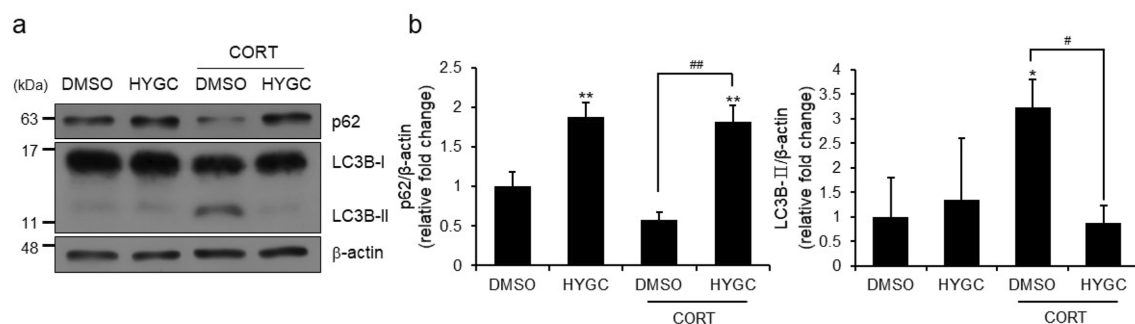


Fig. 5 Inhibitory effects of HYGC on CORT-induced autophagy in PC12 cells. **a**, **b** PC12 cells were treated with HYGC (25 μM) or DMSO (vehicle control) in the absence or presence of CORT (400 μM) for 12 h. Autophagy-related gene expression was measured by immunoblotting. β-actin was used as a loading control. **a** Representative image of autophagy-related protein levels. **b** Quantitative analysis of protein levels. Data were analyzed using two-way ANOVA followed by Tukey's post hoc test. (Mean ± SD, n = 3; $*p < 0.05$ and $**p < 0.01$ compared to the DMSO control group; $^{##}p < 0.01$ compared to the group treated with CORT only)

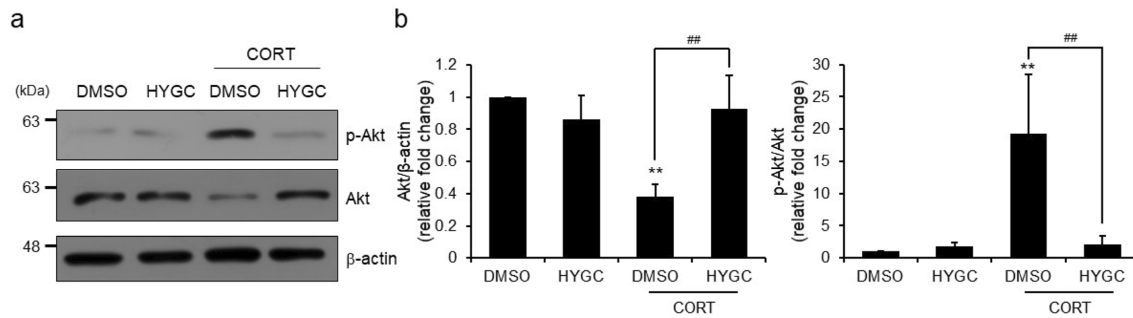


Fig. 6 Recovery effects of HYGC on the expression of Akt proteins induced by CORT. **a**, **b** PC12 cells were treated with HYGC (25 µM) or DMSO (vehicle control) in the absence or presence of CORT (400 µM) for 12 h. Protein levels of phosphorylated and nonphosphorylated forms of Akt were measured by immunoblotting. β-Actin was used as a loading control. **a** Representative images of protein levels. **b** Quantitative analysis of protein levels. Data were analyzed using two-way ANOVA followed by Tukey's post hoc test. (Mean ± SD, n = 3; $^{*}p < 0.05$ and $^{**}p < 0.01$ compared to the DMSO control group; $^{\#\#}p < 0.01$ compared to the group treated with CORT only)

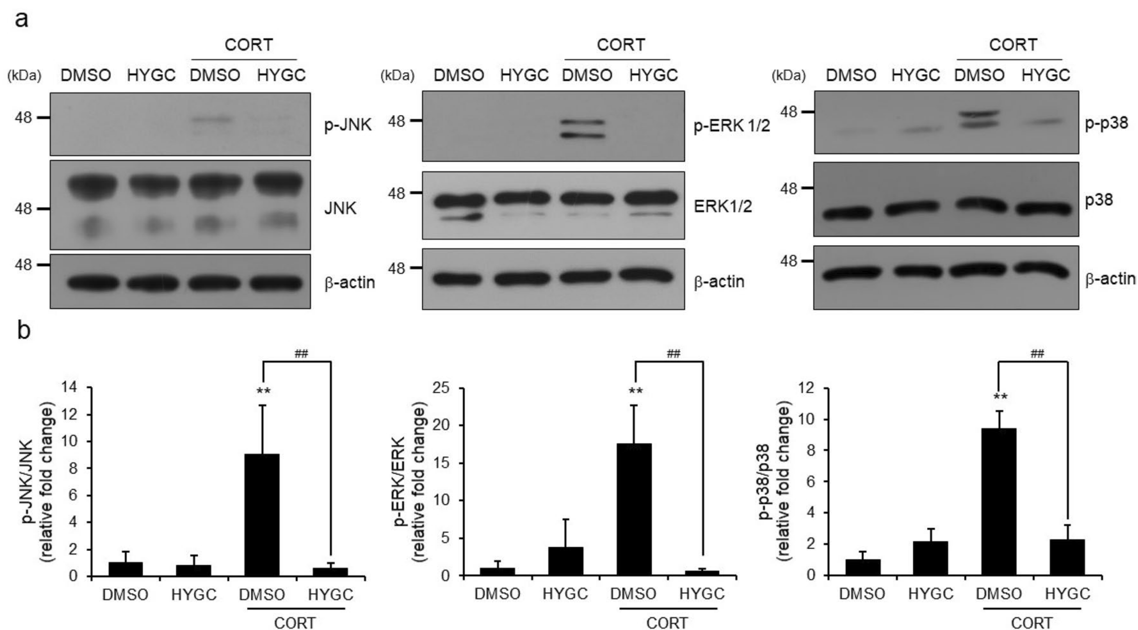


Fig. 7 Effects of HYGC on CORT-induced MAPK activation in PC12 cells. PC12 cells were treated with HYGC (25 µM) or DMSO (vehicle control) in the absence or presence of CORT (400 µM) for 12 h. Protein levels of phosphorylated and nonphosphorylated forms of MAPK were measured by immunoblotting. β-Actin was used as a loading control. **a** Representative images of protein levels. **b** Quantitative analysis of protein levels. Data were analyzed using two-way ANOVA followed by Tukey's post hoc test. (Mean ± SD, n = 3; $^{*}p < 0.05$ and $^{**}p < 0.01$ compared to the DMSO control group; $^{\#\#}p < 0.01$ compared to the group treated with CORT only)