CORRECTION

ERRATUM: Author-Reported Correction of Western Blot Data: Kim JH, Park B, Gupta SC, Kannappan R, Sung B, and Aggarwal BB. *Antioxid Redox Signal* 16:413–427, 2012

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Abstract

This is a non-peer-reviewed author-reported erratum addressing that Zyflamend sensitizes tumor cells to TRAIL-induced apoptosis through upregulation of death receptors and downregulation of survival proteins: role of reactive oxygen species-dependent CCAAT/enhancer-binding protein-homologous protein pathway. Kim JH, Park B, Gupta SC, Kannappan R, Sung B, and Aggarwal BB. *Antioxid Redox Signal* 16:413–427, 2012. The authors claim that Figure 7 reporting Western blot data was erroneous. Specifically, the β -actin panel of Fig. 7B was found to be switched with that of Fig. 7D. The corrected version is reported here. The authors claim that this correction does not influence the conclusion of the study.

THIS IS A request for correction addressing that Zyflamend sensitizes tumor cells to TRAIL-induced apoptosis through upregulation of death receptors and downregulation of survival proteins: role of reactive oxygen species-dependent CCAAT/enhancer-binding protein-homologous protein pathway. Kim JH, Park B, Gupta SC, Kannappan R, Sung B, and Aggarwal BB. *Antioxid Redox Signal* 16:413–427, 2012.

On behalf of all authors, I report that the β -actin panel of Fig. 7B was found to be switched with that of Fig. 7D in the original publication. The corrected panels are shown in the current illustration.

The authors claim that this correction does not influence the conclusion of the study.

The authors regret this error and apologize for any confusion or inconvenience it may have caused.

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FIG. 7. Zyflamend-induced upregulation of DR5 and CHOP is mediated through generation of reactive oxygen species (ROS). (A-B) HCT-116 cells were treated with indicated concentrations of *N*-acetyl-L-cysteine (NAC) (A) or GSH (B) for 1 h, and then treated with 250 μ g/mL Zyflamend for 24 h. Whole-cell extracts were prepared and analyzed by Western blotting using indicated antibodies. β -Actin was used as a loading control. (C-D) Zyflamend-modulated antiapoptotic and proapoptotic protein expression is dependent on ROS. HCT-116 cells were pretreated with NAC or GSH for 1 h and then treated with Zyflamend (250 μ g/mL) for 24 h. Whole-cell extracts were prepared and analyzed by Western blotting using the relevant antibodies. β -Actin was used as an internal control to verify equal protein loading. (E) Zyflamend-induced DR5 and CHOP expression in pancreatic cancer cells and human embryonic kidney cells is mediated through ROS. Cells were first treated with NAC or GSH for 1 h and then with 250 μ g/mL Zyflamend for 24 h. Whole-cell extracts were prepared and analyzed by Western blotting using indicated antibodies. β -Actin was used as a loading control. The revised panels (B) & (D) are shown above. Beta-actin control for panel (C) and panel (D) was derived from the same gel.